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Archives of Neurology and Psychiatry

Vol. 7 MARCH, 1922 No. 3

A RECONSTRUCTION COURSE IN THE FUNCTIONAL ANATOMY OF THE NERVOUS SYSTEM *

ADOLF MEYER, M.D., AND LOUIS HAUSMAN, M.D.

BALTIMORE

The method to be described has been gradually adapted to the needs and demands of physicians and medical students alike, both groping for a clearer and more plastic conception of the nervous system. The underlying principles have been embodied in a course in neuro-anatomy which translates all facts into three dimensions and so incorporates ontogenetic and phylogenetic studies as to help the worker mold his data in a perspective which is thoroughly dynamic. The result is a sound, practical and tangible working basis for the application of the facts as they are experienced.

Since 1893, when Prof. Donaldson granted him the privilege of conducting an elective course in the comparative anatomy of the nervous system at the University of Chicago, and two years later also the regular course in brain anatomy at that institution, Dr. Meyer has had the opportunity from time to time of giving students of greatly varied preparation a review of the essentials and methods of study of the nervous system. The first printed statement appeared in 1898 as part of a review [1] of the neuron doctrine. In it Dr. Meyer presented his plan of the nervous system, which was the outgrowth of his first researches on the forebrain of reptiles and his study of the work of the great English neurologist, Hughlings Jackson, who, in the formulation of his three-level theory, shaped a new and bigger perspective for the neurologist's needs.

Dr. Meyer's duties at the New York Institute forced him to formulate a course for State Hospital physicians, in which an attempt was made to embrace the method of glass-plate reconstructions and the use of plasteline, so as to render the facts convertible into three dimensions,

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* Read at the Forty-Seventh Annual Meeting of the American Neurological Association, Atlantic City, N. J., June, 1921.

* From Phipps Clinic, Johns Hopkins Hospital.

1. Meyer, Adolf: Critical Review of the Data and General Methods and Deductions of Modern Neurology, J. Comp. Neurol. **8**:3-4, 1898.

thereby insuring the acquisition of data in a controllable form. In the meantime, Dr. Oliver Strong used these conceptions in his courses laid down in Bailey's "Text-Book of Histology." Finally, at the Johns Hopkins Medical School, the opportunity presented itself of reaching medical students and graduate workers who were willing to lay their anatomic foundations in this way. During this last year two groups, one of fifty second-year and another of sixty-three first-year students, called for this elective work, and, with the collaboration of Dr. Louis Hausman, the course was given the form we wish to sketch here. Each student made a model—the only way to offer a basis of discussion of difficulties or errors. Each course covered two forenoons a week for a trimester, with one-half to one hour devoted to conjoint discussion and demonstration and the rest to individual work on the models and drawings.

The underlying pedagogic principle is not to allow the worker to think of, talk of or work with, anything that is not clearly established and connected in his own experience. It embraces the data and methods of comparative anatomy, embryology, physiology and experimental and pathologic anatomy. It utilizes nature's experiments in the study of the secondary and experimental degenerations, culminating in a conception of the integration of the nervous system, which, at all times, is readily adaptable to the needs of any situation. By getting away from a burdensome terminology and from endless detail, and by teaching him to work unyieldingly with the facts he can grasp and put together into a practical whole, the student is emancipated from the chaotic and bewildering task usually forced on him, as he is required to combine and survive the diverse teachings of the different courses in his training.

An element of pedagogic philosophy underlies the principle. At no time is the student permitted to lose the view of the whole living biologic organism. Facts and functions must be the issue of the course, not words. Books and written instructions are avoided. Actual material of embryology and comparative neurology furnish all the schemata that are needed; all other schemata are taboo. We begin at once with the individual as we see him and review from head to foot the anatomic parts, as they have to work together in groups; thus is embraced a rational system of segmental or reflex units which the nervous system has to integrate into a personality.

The body is readily viewed as a series of transverse body-segments, held together by a neural tube and ganglion chain, with the following longitudinal arrangement: (1) ventrally the motor-plate, (2) dorsolaterally the receptor-plate, and (3) most dorsally—the roof structures furnishing the later acquisitions. Thus we find a reasonably correct

and simple scheme of transverse and longitudinal groupings, forming a division of the nervous system into two essential mechanisms: segmental and suprasegmental; the former with its intrasegmental and intersegmental components constitute the basis for all reflex activity, whereas the latter, being a later and more specialized development of the intersegmental provisions, meets the needs of economy by centralizing the equilibratory (both static and kinetic) and affective functions, and also the psychobiologic functions, that is, the working together of our cognitive and intellectual resources and the activities largely of the striped muscle system—the whole making up the personality.

By keeping before our eyes the segments of the body and the transverse and longitudinal subdivisions of the neural tube, we obtain an easily remembered rack on which to hang all other data (Fig. 1).

We first consider the divisions of the neural tube and ganglions in the His-models (Fig. 4) and observe the enlargements and transverse constrictions of the tube, giving us the cord, the HB (hindbrain), MB (midbrain) and FB (forebrain). Similarly we recognize in a longitudinal plane (Figs. 2 and 3):

1. The motor or effector plate with its roots, extending to the cephalic end of the MB.

2. The receptor plate and its roots.

3. The dorsal structures, developing the roof membranes and suprasegmental mechanisms.

Then, turning to the body, we group the muscles and viscera and the sense organs into segments (Fig. 1):

1. The olfactory apparatus.

2. The eye socket and its contents.

3. The jaws forming the mastication and gasserian segment, with its peripheral and central distributions.

4. The ear with its vestibular and cochlear apparatus and the intermediate-facial-abducens group.

5. The taste, deglutition, articulation and thoracic and abdominal visceral segment.

6. The cervical, thoracic, lumbar, sacral and coccygeal segments.

Having recognized these divisions of the body and neural tube, the next logical step is to correlate them (Fig. 1): The olfactory and optic mechanisms form the foundation of the forebrain and midbrain; the isthmus corresponds in the face to the lower orbital edge, marking roughly the limit between the midbrain and hindbrain; the mastication and the auditory-facial-abducens and the taste-deglutition-respiration-articulation segments, respectively, form the anterior, middle and pos-

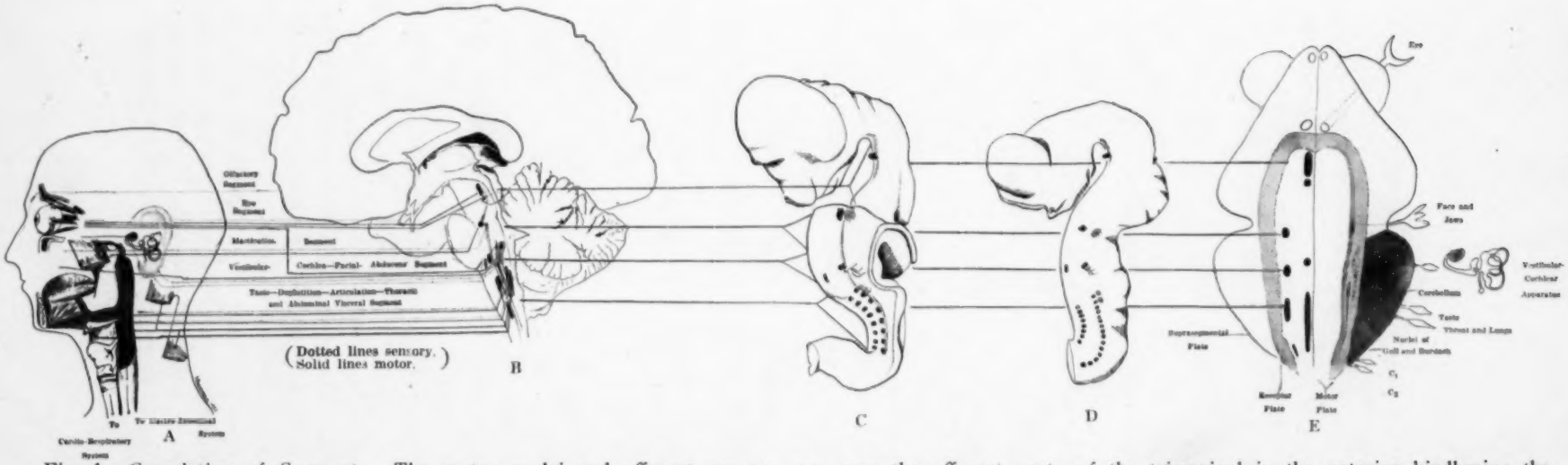

Fig. 1.—*Correlation of Segments.* The motor nuclei and efferent nerves are in solid black; the receptor nuclei are dotted and the afferent nerves outlined.

A, the segments as they can be read off from the body. *B*, median sagittal section of an adult brain with the nuclei of the midbrain and hindbrain represented on one plane. *C*, left lateral view of the brain of a human embryo about 5 weeks old (13.6 mm.), after a His model. The segmental arrangement is very apparent. The motor and receptor plates are well defined and separated by a slight groove in the hindbrain. The motor plate mesially presents the oculomotor nerve in the midbrain—the trochlear nerve appearing dorsally after decussating in the roof, the abducens in the middle hindbrain and the hypoglossal in the posterior hindbrain segment; laterally the trigeminal in the anterior hindbrain, the facial in the middle hindbrain and the glossopharyngeal, vagus and spinal accessory roots in the posterior hindbrain. In the receptor plate are seen the afferent roots of the trigeminal in the anterior hindbrain; the nervus intermedius (or the afferent fibers of the facial nerve) and the vestibular and cochlear nerves in the middle hindbrain, and the afferent fibers of the glossopharyngeal and vagus nerves in the posterior hindbrain. *D*, brain of human embryo (10.2 mm.) after a His model. The segmentation is even more clearly defined than in *C*, due to the fewer bendings of the neural tube. *E*, primitive neural tube opened up and spread out on one plane. Situated mesially in the motor plate from above down are the oculomotor, trochlear, abducens and hypoglossal cell clusters or nuclei; laterally, the trigeminal or mastication, facial and ambiguus nuclei. The receptor plate presents the trigeminal complex, the tractus solitarius with its nucleus, and the vestibular cell-mass.

terior hindbrain segments and the foundation for the cerebellar apparatus; the cord segments are clearly marked by roots and intercostal and equivalent subdivisions of the body (Fig. 3).

This correlation is further enhanced and simplified if we study the neural tube from the inside out, that is, from the ventricle outward. Thus we are readily enabled to establish a direct relation between the

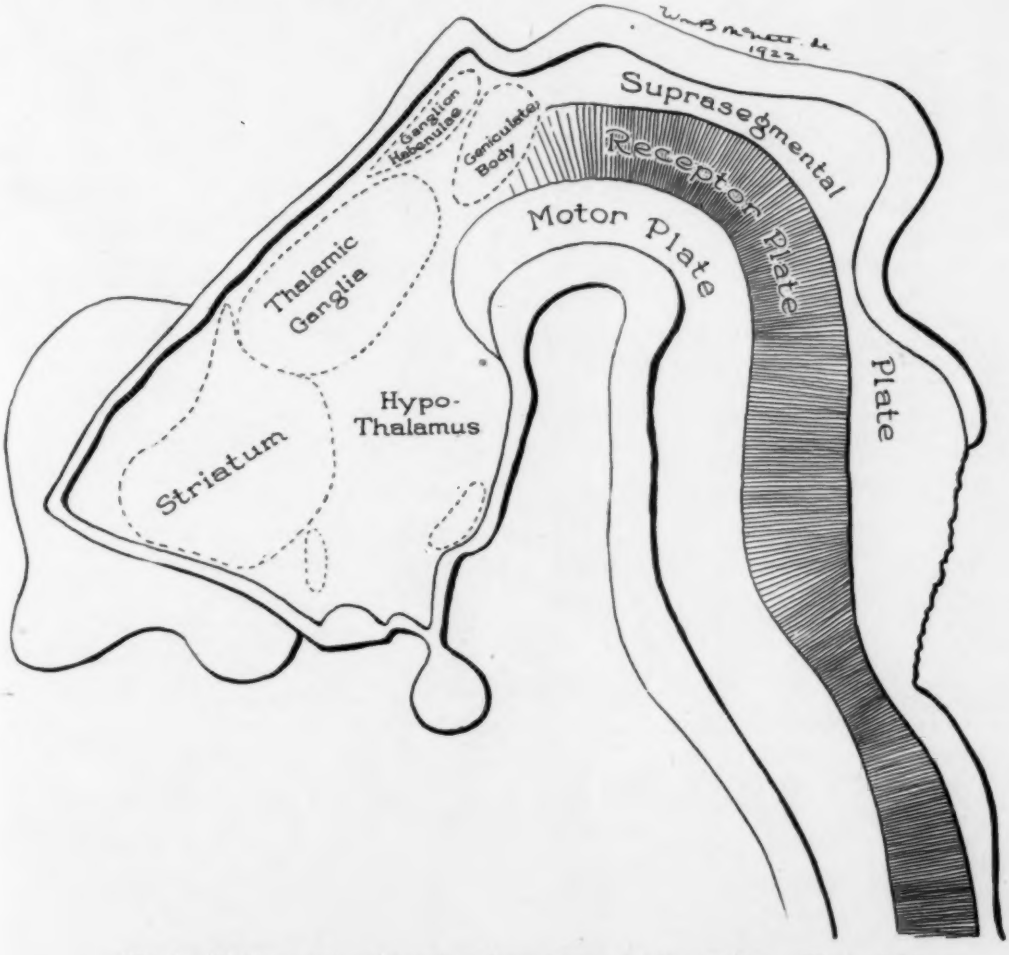

Fig. 2.—Median section through embryo human brain. (After a His model.)

neural tube and what we see in every living patient—a relation to muscle groups and viscera and sense organs and sensory surfaces. Each segment of muscles and glands has its efferent or effector provisions which are played on by definite afferent elements, connected with segmentally arranged sense organs; these effector and receptor ele-

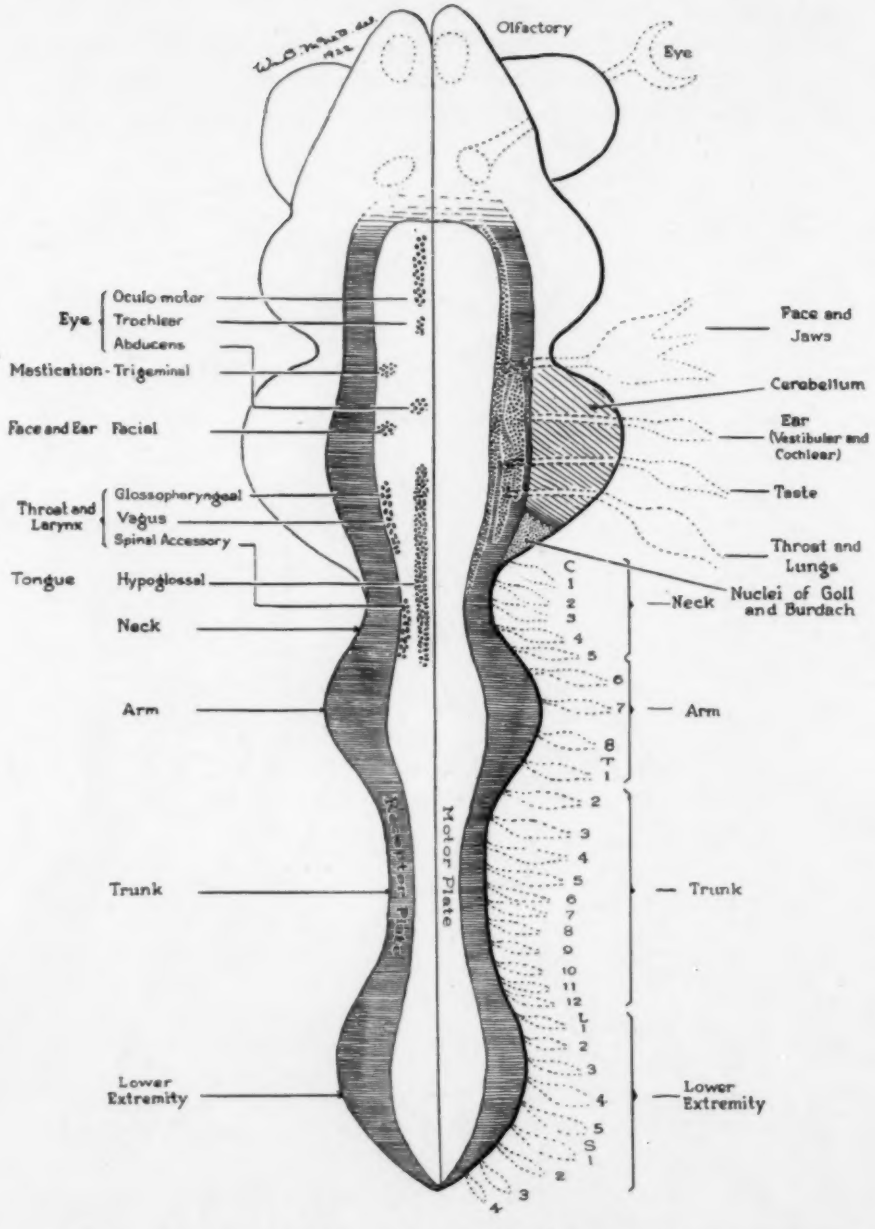

Fig. 3.—Scheme of primitive neural tube opened up, with the various parts represented on one plane.

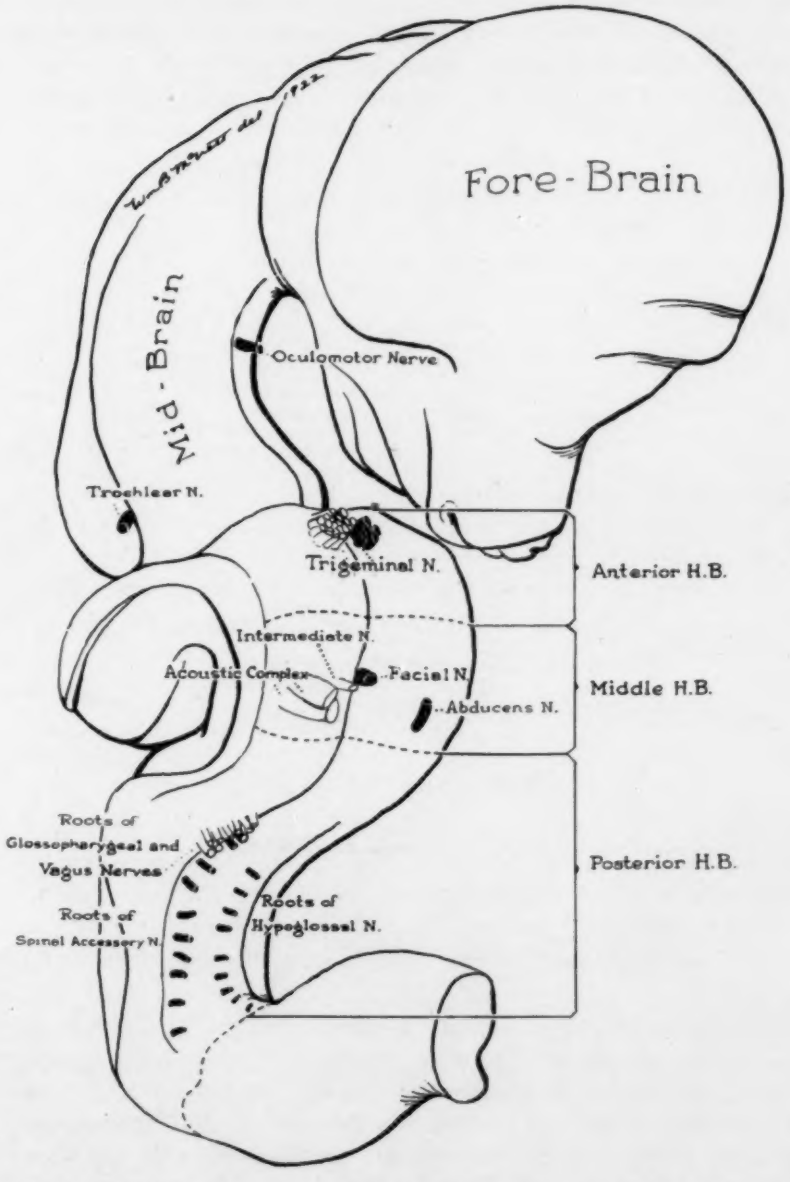

Fig. 4.—Brain of a human embryo 5 weeks old (13.6 mm). (After a His model.)

ments, which the neural tube presents, are in turn coordinated by intrasegmental and intersegmental components, the ultimate integration of these various parts being achieved by the suprasegmental cerebellar and cerebral mechanisms. Whatever is added to the simple segmental stage grows on the outside (Fig. 5). That is why we begin with the simple neural tube and work from the ventricle outward, instead of beginning with the "pons" and other late surface additions and working backward; the former approach is simpler and more logical. Therefore we start with the segmental parts. The open tube (Fig. 3) gives the fundamental scheme.

We resolve the tube into its various elemental mechanisms by experimental analysis and to that purpose employ the Gudden, Nissl and Wallerian types of degeneration of the peripheral or root elements to establish the actual segmental mechanisms. Further analysis identifies the ground bundles and reticular substance of the tube as the intersegmental elements. The dorsal excrescences or terminal bulgings of the tube form the suprasegmental organs: (*a*) the cerebellar organ; (*b*) the midbrain roof organs, and (*c*) the forebrain.

Extirpation of a suprasegmental organ singles out in each segment (*a*) the cells of the suprasegmental afferent neurons by the axonal reaction and atrophy, and (*b*) the suprasegmental efferent fibers by outgoing degeneration. These experimental principles are always kept in view, since they give us the objective definition of what we work with. For the reconstruction of the segmental apparatus the reader should consult Figures 6 to 9, inclusive.

SUPRASEGMENTAL ORGANS

As we ascend the vertebrate scale, we find that the pattern of the neural tube becomes more and more differentiated; the nervous architecture takes on various designs; we note that the differentiation runs parallel with the behavior resources of the organism as we get further away from the simple reflex type of the more or less segmental animal.

In the process of evolution, economy develops specialized organs, that is, the suprasegmental structure for the centralizing of definite types of function of the segmental animal. The lack of such economy and centralization would provide a system entirely too cumbersome for the needs of man for prompt activity. In the amphioxus, for example, there are, according to Kölliker,[2] twelve large cells in the cranial part sending fibers to the more caudal segments, and fourteen similar cells in the caudal segments sending their fibers to the cranial part. For the complex correlations in man, innumerable connections of this type

2. Kölliker: Handbuch der Gewebelehre des Menschen, 1893.

would be needed between the head, neck, trunk and lumbosacral segments; the tube would be encumbered and the time of interaction too slow. Instead of this pattern, we find added to the simple reflex segmental components, the suprasegmental structures:

1. The cerebellar station with its afferent elements from the segments and efferent paths carrying to these segments the net result of

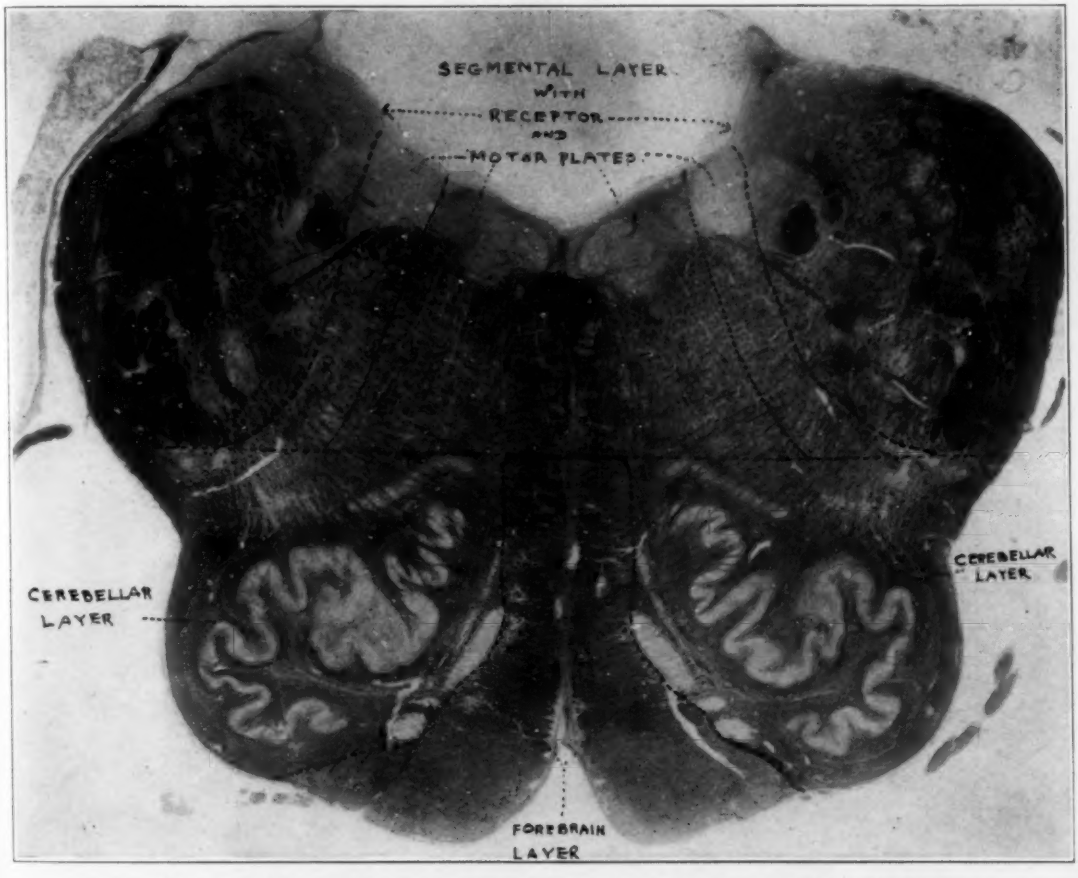

Fig. 5.—Photograph of a cross-section through the posterior hindbrain segment of an adult brain, showing from within out—the segmental layer with its motor and receptor plates, the cerebellar layer and finally the forebrain layer with its afferent and efferent components. (Weigert stain.)

cerebellar synthesis—that is, the elaboration of all the equilibration and balancing functions of motility.

2. The colliculi, belonging to the midbrain and highly developed in the lower animals, with their afferent and efferent mechanisms.

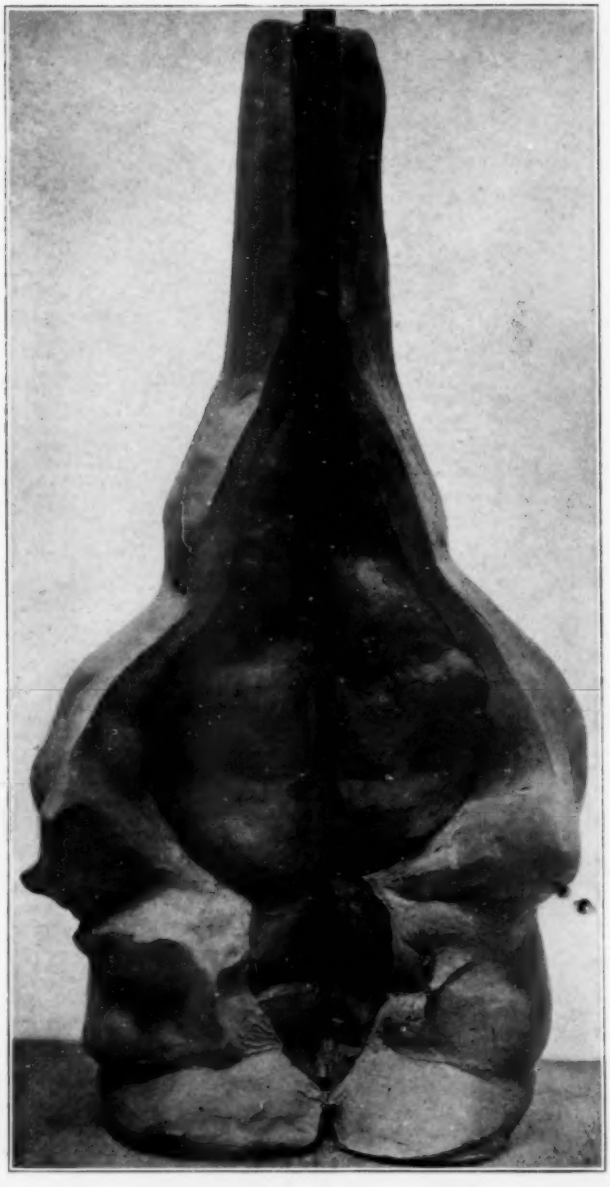

Fig. 6.—*Dorsal View.* Reconstruction of the segmental plates of the midbrain and hindbrain, viewed from within the hindbrain ventricle, at the base of which the spinal canal is seen opening into it. Note the marked narrowing of the plates toward the cephalic limit of the midbrain and the extent to which the receptor plates overlap the motor. Owing to the marked flare of the receptor plates in the middle hindbrain segment, the ventricle is broadest at that level. Immediately below this flare, on either side, are seen the projections for the attachment of part of the choroid plexus. Reduced from photograph × 4.7.

3. The forebrain apparatus, for the coordination and central elaboration of all the conditions of psychobiologic functions.

Cerebellum.—Embryology shows us how and where to add the cerebellar organ. This suprasegmental structure, developing dorsally over the vestibular and trigeminal segments, consists of a terminal

Fig. 7.—*Ventral View of Figure 6.* The motor and receptor plates, showing the additional reconstruction of the nuclei which appear on the ventral surface of the former. In the mesial part of the midbrain, the motor plate, containing the oculomotor and trochlear nuclei, forms a beak-like projection which gradually disappears in the anterior hindbrain segment. In the lateral part of the motor plate, we recognize, from above down, the trigeminal or mastication nucleus in the anterior hindbrain segment, the facial nucleus in the middle hindbrain segment and the slender ambiguus nucleus for the glossopharyngeal, vagus and accessory nerves in the posterior hindbrain segment. The ventral horns at the base mark the transition of the motor plate of the spinal cord into that of the hindbrain; × 2.8.

Fig. 8.—*Dorsal View.* Reconstruction of the segmental cell groups appearing in the dorsal part of the motor and receptor plates.

The motor plate presents mesially from above down the round abducens nuclei in the middle hindbrain and the slender hypoglossal nuclei occupying the greater part of the posterior hindbrain segment, in the lateral portion of which is seen the dorsal preganglionic sympathetic nucleus, lying adjacent to the receptor plate.

The right receptor plate presents the large vestibular cell-mass in its entire length and the cochlear complex which juts off perpendicularly to allow sufficient space for the restiform body to pass through (Fig. 15).

In the left receptor plate, the caudal portion of the vestibular mass has been cut away to show the course of the elongated tractus solitarius.

The groove in the anterior hindbrain and midbrain segments indicates the path of the mesencephalic root of the trigeminal, which can be seen more clearly in Figure 10.

The descending or spinal root of the trigeminal being situated in the ventral part of the receptor plate adjacent to the motor (Fig. 11) is not seen in this figure; × 2.6.

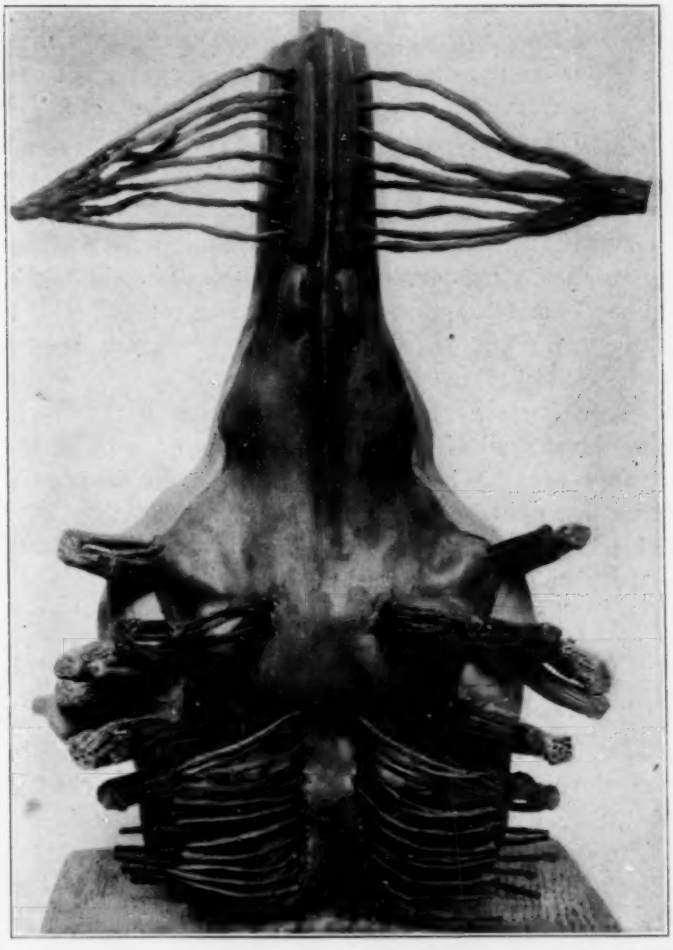

Fig. 9.—*Ventral View of the Segmental Nerve Roots.* The motor plate presents from above down and in the midline the nerve roots of the oculomotor nerves in the midbrain, the abducens nerves in the middle hindbrain and the numerous hypoglossal filaments in the posterior hindbrain segment, occupying the greater part of its length; laterally appear the trigeminal roots of the mastication nucleus in the anterior hindbrain, the facial roots in the middle hindbrain, and the glossopharyngeal, vagus and accessory fibers in the posterior hindbrain segment. The receptor plates present in the anterior hindbrain the afferent roots of the trigeminal, in the middle hindbrain the nervus intermedius and the vestibular and cochlear nerves, and in the posterior hindbrain segment the afferent components of the glossopharyngeal and vagus nerves.

The trochlear nerve by reason of the unique course it follows in passing through the substance of the segmental plates to decussate dorsally in the anterior medullary velum is not shown in this stage of the reconstruction, but can be seen in Figure 16, emerging beneath the inferior colliculus. × 3.

sheet (of cortex) and central ganglions. The pons mass and the inferior olives, which are so intimately associated with it, have wandered from the lip structure to the ventral side of the motor plate (Fig. 5). Thus we have these parts: arising over the vestibular cell-mass the "inner cerebellar arm" with the roof nucleus (Fig. 10); outside of this fundamental arch the dentate nuclei (Figs. 11 and 12) giving off the superior cerebellar arms, and the intermediate nuclei (globosus and emboliformis) lying between the dentate and inner arm (Figs. 11 and 15); the restiform bodies swinging around the dentates, conveying from the segments the bulk of the afferent fibers to the vermis (Figs. 14 and 16), that is, the cerebellar cortex. This terminal sheet of the cerebellum consists primarily of vermis and flocculus (Fig. 15) and later of the more recently developed cerebellar hemispheres, which are reached by the pons arms; that the latter are the most superficial of the cerebellar peduncles, coincides with the fact that they are the last to develop, their development running parallel with that of the opposite cerebral hemisphere.

This sketch of the cerebellar apparatus gives the rack within which to study the histologic details and the results of genetic and experimental work. The elimination of the cerebellum in the young leads to atrophy of all the cerebellar afferent cells and fibers united in the restiform complex: the Clarke column cells and direct spinocerebellar tract, fibers from the posterior column nuclei, and the fibers from the opposite inferior olive (Figs. 13, 14 and 16). Fibers from the lumbar region form the ventral cerebellar tract swinging around the trigeminal root and thence over the superior cerebellar arm to the vermis (Fig. 16).

The cerebello-segmental impulses have several outlets. One set is affected by the cerebellar cortex activity through the roof nuclei and the inner arm, finally reaching the segments through the vestibulo-ocular or vestibulo-cephalic tract (the mesial longitudinal fasciculus) and the vestibulo-spinal tracts (Figs. 11, 13 and 15). Another outlet is formed by the superior arm to the opposite red nucleus, influencing the segments through the rubrospinal pathway (Figs. 17 and 18), and the cerebral basal and cortical mechanisms through the rubrothalamic elements. A third cerebellar efferent path is the hook bundle to the tegmentum of the hindbrain.

The great variations of the cerebellar mechanisms in the animal series make it obvious that the term "pons" should not be used for the designation of the tegmentum and the segments of this region. It refers purely to the neocerebro-neocerebellar connection which forms the ventral superficial protruberance. All these parts are built on the

outside of the original segmental parts. The reconstruction of the cerebellar apparatus is presented in Figures 10 to 18 inclusive.

Midbrain.—The midbrain, originally the dominant integrator of all receptor functions, presents in its roof the auditory colliculi and the optic colliculi resting on the midbrain nucleus of the trigeminus (Figs. 14, 15 and 16). The auditory or inferior colliculus receives the lateral fillet, which is the upward continuation of the trapezium or auditory decussation (Figs. 17 and 18). The optic or superior colliculus in-

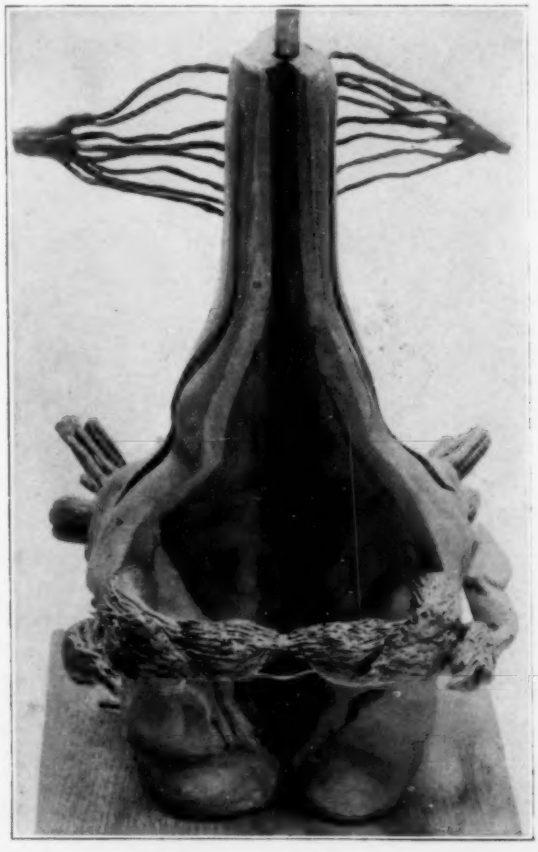

Fig. 10.—*Dorsal View Slightly from Above.* Fundamental arch of the cerebellar reconstruction, showing the inner cerebellar arm arising from the vestibular mass on either side to terminate in the cells of the mesially placed roof nuclei. Resting on the inner arm and lateral to the roof nuclei are the intermediate cell groups (globosus and emboliformis); these parts are also to be seen in Figures 11, 12 and 15.

The efferent mechanisms associated with the inner cerebellar arm (the posterior longitudinal bundles and the vestibulospinal systems) can best be seen in Figures 11 and 13. × 2.5.

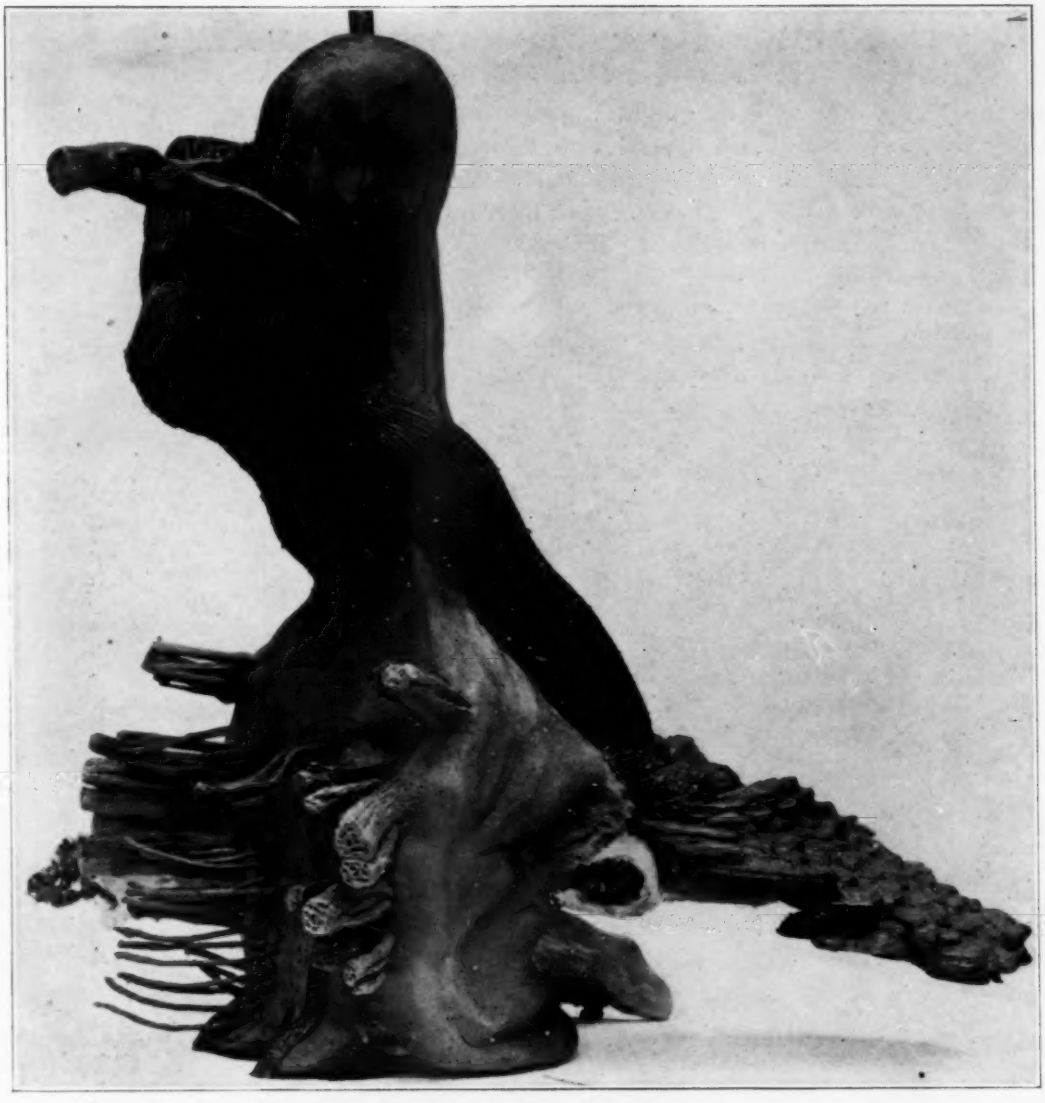

Fig. 11.—*Left Ventrolateral View.* Reconstruction of the superior cerebellar arm, showing the cell-mass of origin (dentate nucleus) and the course of the cell processes as they pass toward the midbrain to decussate (at the level of the trochlear nuclei and ventral to the motor plate) before entering the red nucleus, which has been reconstructed only on the left side of the model; the fibers entering the red nucleus form a distinct shell, which is quite thick on the ventromesial surface (Fig. 15).

Note the posterior or mesial longitudinal bundles which are seen only on the upper part of the motor plate; in the hindbrain they occupy a dorsomedian position and therefore are not seen in this part of the reconstruction. In the lower part of the motor plate the vestibulospinal systems are readily recognized. × 3.2.

fluences the lower segments of the neural tube through the colliculospinal (or predorsal) bundle (Figs. 17 and 18). For the reconstruction of the midbrain consult Figures 14 to 18, inclusive.

Forebrain.—The forebrain is divided into the median line and lateral wall structures, the former presenting the posterior commissure, the pineal recess and pineal gland, the superior or habenular commissure, the taenia and roof membrane with choroid plexus, the

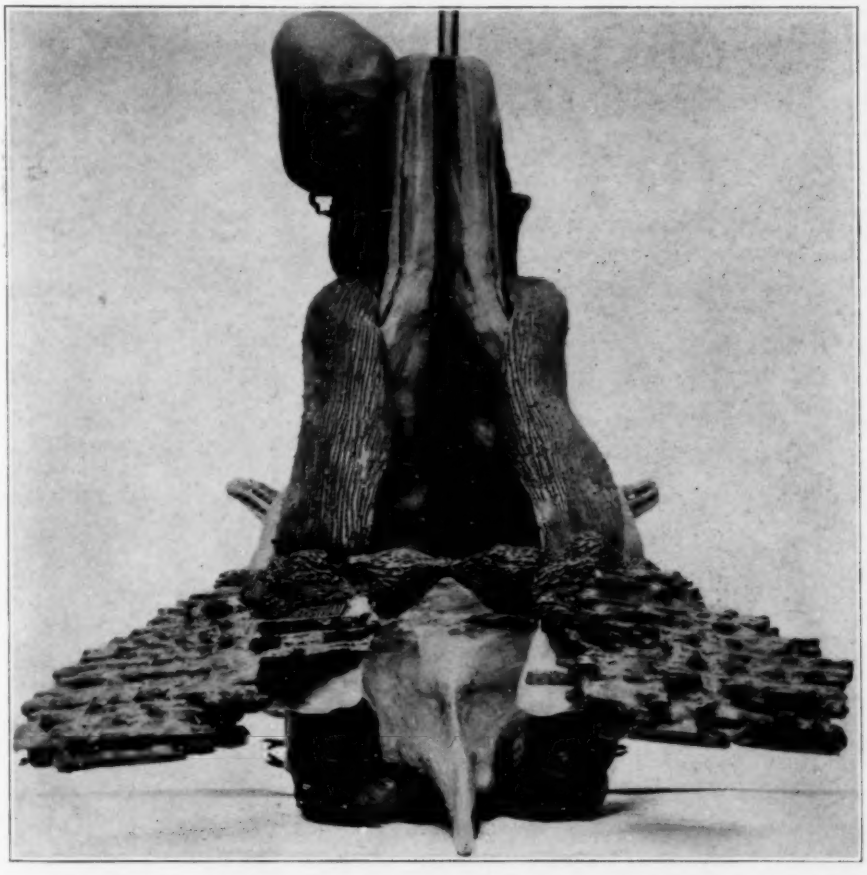

Fig. 12.—*Dorsal View of Figure 11.* The posterior medullary velum (in white) has been added. ×2.5.

neuropore region, anterior commissure, lamina terminalis, optic recess and chiasm, infundibulum and pituitary body, tuber cinereum and saccus vasculosus region, and the mamillary region and interpeduncular ganglion. The lateral walls consist of the thalami (with the obliterated eye protrusion) and the olfactory protrusion, carrying also the striatum and the hemisphere cortex.

The construction of the forebrain rests on the midbrain and brings out:

1. The hypothalamus and floor of the median forebrain ventricle (or third ventricle), with the infundibulum, tuber cinereum, corpus mammillare, the hypothalamic nucleus (corpus Luysii) and the substantia nigra.

Fig. 13.—*Ventral View.* Beginning of the reconstruction of the inferior cerebellar arm or restiform body. The inferior olives with the interolivary and olivocerebellar fibers are shown. Note the relation of the hypoglossal roots to the olives. On the right, immediately adjacent to the acoustic and glossopharyngeal nerves, the lateral pocket with its choroid plexus is seen. × 3.1.

2. The thalamus thickness proper: the anterior, median, lateral, dorsal, central and posterior nuclei (pulvinar and geniculate bodies), and continued posteriorly, the red nucleus.

3. The epithalamus: taenia, ganglion habenulae, superior commissure and the retroflex bundle to the interpeduncular ganglion, which in turn is connected with the central gray of the anterior hindbrain segment.

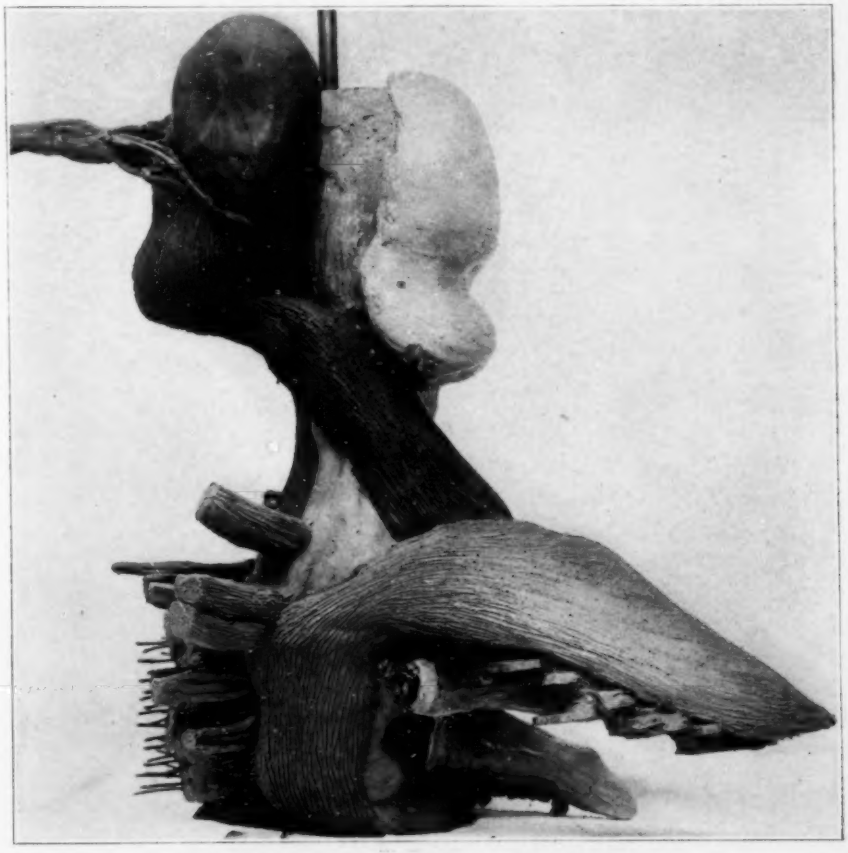

Fig. 14.—*Left Lateral View of Figure 13.* The direct cerebellar tract is seen flowing into the restiform body, which sweeps in broad curves over the superior cerebellar arm and the dentate nucleus. The superior and inferior colliculi have been added, preparatory to the construction of the anterior medullary velum and the cerebellar cortex (Fig. 16). Note the left trochlear nerve appearing between the inferior colliculus and the superior cerebellar arm.

In this figure the tuberculum acusticum has been left off in order not to obscure the part of the restiform body which it embraces; these relations can be seen in Figure 15. The lateral pocket has been cut off. ×2.5.

Having constructed these parts, we next place the optic chiasm and the optic tracts, which should be put on loose enough to let the crus cerebri (to be constructed later) pass to the basal surface. Above the chiasm we build the lamina terminalis, carrying dorsally the anterior commissure which connects the striata and the olfactory bulbs. In relation to this phase of the reconstruction, we consider the paraterminal body, consisting of the anterior perforated substance, with the

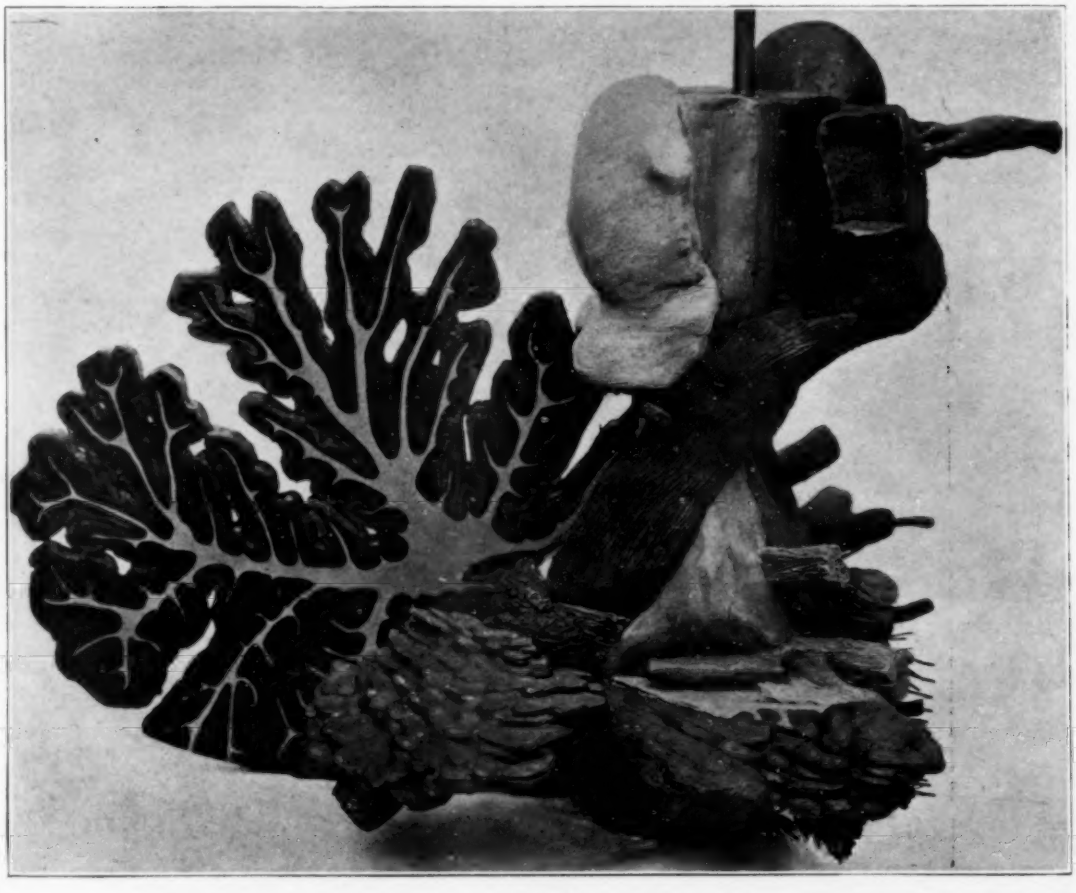

Fig. 15.—*Right Lateral View.* Note the addition of the cerebellar cortex (only a slice through the mesial part of the vermis being indicated) and the flocculus with its peduncle. The restiform body, embraced by the cochlear mass, is cut off in order not to obscure the other structures; reduced from photograph × 3.3.

pedunculus septi and its nucleus basalis, and the commissural mass—corpus callosum, septum pellucidum, fornix or hippocampal commissure and fornix column.

The next stage of the reconstruction comprises the olfactory lobe and tubercle, the lateral olfactory root and gyrus ending in the cortex of the amygdala and uncus, and the mesial olfactory gyrus, phylogenetically the first and oldest part of the highest integrator—the cerebral cortex.

Over the amygdala there arises the corpus striatum, perforated by the internal capsule, which anteriorly, and behind and below, only

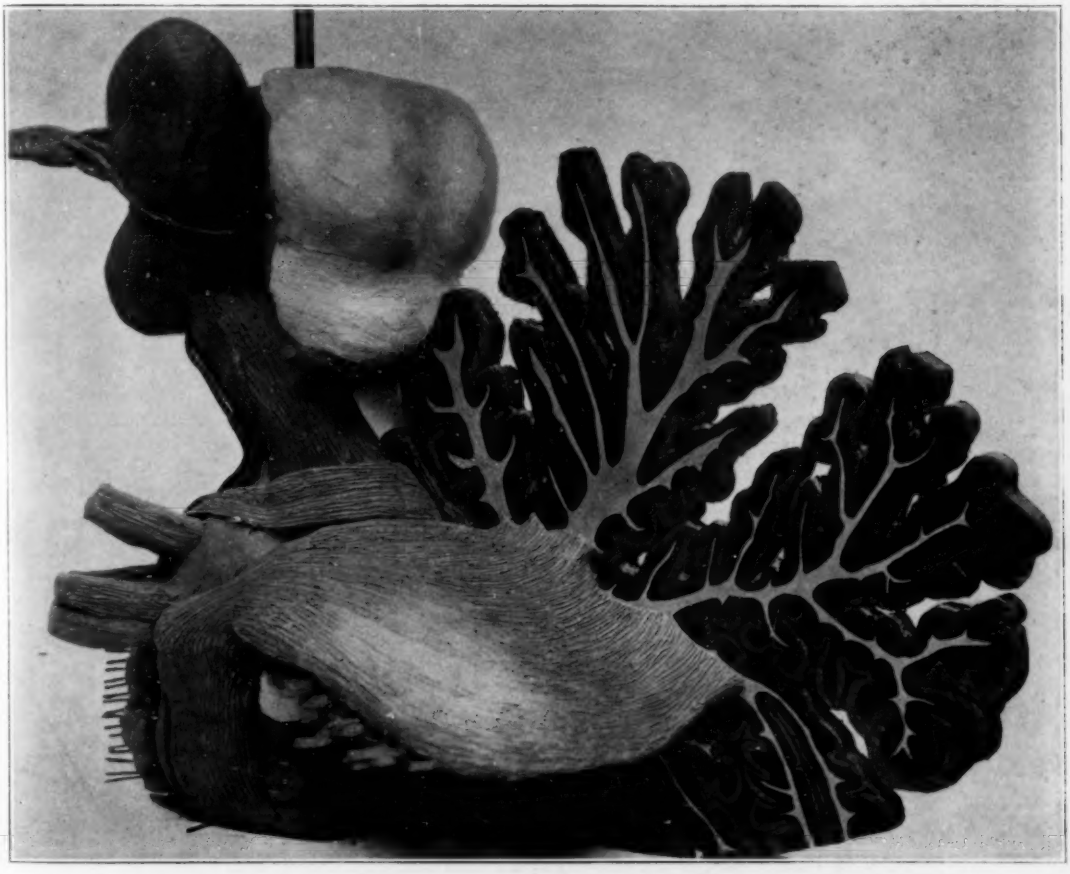

Fig. 16.—*Left Lateral View.* The restiform body is seen entering the cerebellar cortex. The indirect cerebellar tract described by Gowers has been added; after hooking around the trigeminal roots it passes over the superior cerebellar arm to terminate in the cells of the cerebellar cortex; its spinothalamic component, whose course is here only partly indicated, can best be studied in Figure 17. Ventral to the latter is the rubrospinal tract. Reduced from photograph × 3.3.

partly splits off the caudate from the putamen mass. By means of the striato-Luysian bundle, the large-celled mass, that is, the globus pallidus, is connected with the hypothalamus and substantia nigra.

Fig. 17.—*Left Ventrolateral View.* Reconstruction of the superior olives, lateral fillets, thalamo-olivary and colliculospinal systems. The superior olives occupy the lateral portions of the middle and anterior hindbrain segments; the left lateral fillet is seen entering the inferior colliculus of that side, whereas the right, which follows a similar course, is shown only in part; immediately ventral to the posterior longitudinal bundles are the colliculospinal or predorsal fasiculi, the interolivary fibers having been removed in order to give an unobstructed view; between the superior olive and the colliculospinal tract courses the thalamo-olivary bundle, which is seen to spread out over the lateral surface of the inferior olive. Note the rubrospinal tracts and their crossing in the upper part of the superior cerebellar arm decussation. Attention is directed to the course of the spinothalamic tract as it winds beneath the lateral fillet to ascend along its mesial border. To give a better view of the indirect cerebellar tract, the glossopharyngeal, vagus and accessory nerve roots have been removed. × 3.3.

From the olfactory tubercle to the uncus there extends as the dorsomesial border of the hemisphere cortex the mesial olfactory gyrus, consisting of the striae Lancisii and cornu ammonis, the latter also giving origin to the mesial wall or fornix radiation of the hemisphere.

Fig. 18.—*Ventral View* of *Figure* 17, with the addition of the trapezoid body and its nucleus. × 3.2.

Between the mesial olfactory and the lateral olfactory gyri, the convexity of the hemisphere is formed by the neocortex, giving rise to the lateral wall radiation, that is, the internal capsule.

We next add the cerebral afferent tracts consisting of the auditory, visual and general sensory (mesial and superior) fillets and the efferent tracts consisting of the corticopontile and corticosegmental or pyramidal systems.

Thus we find in man essentially three dorsal or dorsolateral blind pockets or terminal bodies of the neural tube, each one a suprasegmental organ: the cerebellum, the striatum and the cerebral cortex. The thalamus is treated as a relay station with some terminal functions, but in the main having definite interdependence with the hemisphere functions. It is the cerebral cortex that brings under one head the psychobiologic integrations.

COMMENT

This bare outline has been set forth with the idea of conveying the perspective we aim to attain in familiarizing the student with the data of neuro-anatomy. Before he is led into a maze of innumerable details, he is first provided with a solid three dimensional foundation and a general frame, in which it becomes comparatively easy for him to insert the discussion of the rubral system or any other special points. It gives him a dynamic concept and a plastic instrument to work with, and every step, building from the inside out, first the segmental and then the suprasegmental structures, is determined in gross specimens and models, serial sections and photographs, and brought to expression in a plasteline reconstruction.

GUIDE TO FIGURES

Figures 6 to 18, inclusive, are photographs of an accurate reconstruction (of the midbrain and hindbrain of an adult) made by Dr. Hausman from a complete series of Weigert-stained cross-sections, each 66 microns thick. The material used is plasteline, which is ideal for this purpose as it provides simplicity without sacrificing accuracy. The original model is enlarged six times, and in colors: blue and brick-red for the receptor and motor segmental elements, respectively, orange for the efferent cerebellar mechanisms and violet for the auditory apparatus.

Figures 6 to 9, inclusive, give the segmental reconstruction; Figures 10 to 16, inclusive, the cerebellar reconstruction with the pons connections left off; Figures 17 and 18, the midbrain.

A model of the forebrain is in process of construction and an article on the forebrain will be published at a later date.

INTRACRANIAL ANEURYSM OF THE VERTEBRAL ARTERY *

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Although small aneurysmal dilatations are common in any and all of the cerebral vessels, large intracranial aneurysms are rare. Thus, Hoffmann,[1] in reporting on seventy-eight intracranial aneurysms observed during twenty years' examination of medicolegal cases in Vienna, describes chiefly minute dilatations or diffuse enlargement of the cerebral arteries, and not aneurysms of a size that would be likely to produce pressure symptoms. Most of these cases had come to necropsy because of sudden death from rupture of the dilatations, and had not been observed clinically. The aneurysms were distributed as follows in seventy-five cases:

Arteries of the sylvian fissures	20
Carotids	13
Anterior communicating	12
Basilar	10
Vertebral	10
Miscellaneous	10
Total	75

Of these, fifty-three were in women and twenty-two in men, which reverses the proportions usually observed in aneurysms. It is to be noted that of the ten aneurysms of the vertebral artery, seven were on the left side; this predominance of the left side is usually described. Thus, Oppe,[2] who in 1892 collected reports of seven cases of aneursyms of the vertebral artery, found that six were in the left.

Möser,[3] describing three cases of what were virtually simple dilatations of the left vertebral artery, comments on the fact that thrombosis and embolism also usually affect the left vertebral artery. He made a study of the vertebral arteries in several bodies and found that the left artery is usually somewhat larger than the right (average, 2.43 as against 2.18 mm. diameter), but that there is almost always a constriction at the point where the left vertebral joins the basilar, and less often on the right side, the average amount of narrowing being 0.318 and 0.24 mm. for the left and right arteries, respectively.

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* Presented by request before the Chicago Neurological Society, Dec. 15, 1921.

* From the Department of Pathology, University of Chicago, and the Otho S. A. Sprague Memorial Institute.

1. Wien. klin. Wchnschr. **7**:824, 1894.
2. München. med. Abhandl. Ser. 2, 1892; quoted by Steckelmacher.
3. Möser: Deutsch. Arch. klin. Med. **35**:418, 1884.

In 142 cases of cerebral aneurysm reviewed by Krey [4] none was in the vertebral arteries. Beadles [5] compiled 555 cases of aneurysm of the cerebral arteries, all but 114 of which had been previously published. Unfortunately, he gives no analysis of this great material, but he has little to say concerning vertebral artery aneurysms, so presumably they were not numerous. He mentions only three such cases. The chief facts brought out by him are that in 46.3 per cent. of cerebral aneurysms the first symptom was apoplexy, in 67 per cent. death was preceded by apoplexy, and in 20.7 per cent. there were clinical evidences of intracranial tumor, which most often occurred with aneurysms of the basilar artery, which he discusses at some length. In only 16.39 per cent. was there no cerebral or meningeal hemorrhage. He describes one aneurysm of the left internal carotid artery which measured 1¾ by 1⅛ (4.5 cm. by 2.9 cm.) inches, and states that this ranks among the largest intracranial aneurysms on record. "Its size has been exceeded by few, probably less than a dozen." These large aneurysms have arisen mostly from either the carotid or the basilar artery; he makes no mention of vertebral aneurysms in this group. Some of these aneurysms were the size of "a small orange," "a duck's egg," "a small hen's egg," "the size of a nut," whatever these comparisons may signify. Some, with measurements given, exceeded 2 inches in diameter. The largest vertebral aneurysm that I have found reported, that of Steckelmacher,[6] is uncertain since the diagnosis rested on clinical evidence only. The aneurysm eroded the left half of the supra-occipital bone, and formed a large visible external swelling. The patient was still alive when the case was reported, so the origin cannot be considered as positively established.

The classical illustration of aneurysm of the vertebral artery published by Cruveilhier [7] in his famous atlas, involved the right vertebral artery, and, to judge from the illustration, measured about 15 mm. in diameter.

There has appeared recently a report of a case of vertebral aneurysm of comparatively large size, described by J. Floyd Morrow.[8] This was observed in a body used for dissection in a medical school, and consequently there was no history.

The man was apparently 50 years of age, and, as no other evident cause for his death was found, it was assumed that he had died from paralysis of

4. Inaug. Dissert. Greiswald quoted by Rhein: J. Nerv. & Ment. Dis. **41**:360, 1914.

5. Beadles: Brain **30**:285, 1907.

6. Steckelmacher: Deutsch. Ztschr. Nervenheilk. **54**:347, 1916.

7. Cruveilhier: Anatomie Pathologique du Corps Humain **2**:28, 1835, pl. 3.

8. Morrow, J. Floyd: Med. Rec. **100**:894, 1921.

the vital centers. The aneurysm is described as "a spontaneously cured fusiform aneurysm of the right vertebral artery. The aneurysmal dilatation began 1.5 cm. from the bifurcation of the basilar artery, and increased abruptly to its greatest diameter of 2.5 cm. This extreme dilatation was continued for a distance of 3 cm. downward and the artery again very abruptly assumed its normal diameter." There was a channel on the side toward the medulla, of approximately normal size. There was much calcification of the wall. The left vertebral artery was crowded laterally and showed a dilatation for about 2.5 cm. of its course with a diameter of nearly 1 cm. This artery was very sclerotic as also was the circle of Willis. The medulla was pressed into an unrecognizable mass by the aneurysm. The anterior surface of the medulla was concave, much flattened and pushed backward between the cerebellar lobes, but there was no softening. The pons was pressed slightly upward on the left. Much degeneration of sensory and motor tracts was shown by Marchi staining. There is no statement made as to evidence of syphilis elsewhere in the body.

Most of the other cases recorded concern small aneurysms, like the "cherry pit" sized aneurysm of the left vertebral artery reported by Schultze,[9] which was unusual in producing a marked convulsive tic involving the left facial nerve, without degeneration of the nerve. In this case death resulted from pulmonary tuberculosis. We also have the two vertebral aneurysms observed among twenty-two cases of ruptured intracranial aneurysms described by Wichern.[10] Both of these were in males, one in the right vertebral artery, 3 to 4 mm. in diameter, and one in the left, "a pea sized sac."

Weidman [11] states that since Beadle's report in 1907 only seven cases have been indexed up to 1915. The largest of these aneurysms was reported by Boinet,[12] measuring 30 by 15 mm., involving the left vertebral artery, and associated with several gummas of the brain and meninges. An aneurysm like that in Wiedman's case also occurred in a man, aged 40, with syphilis, but it was only "pea sized."

A striking feature about most of the ruptured cases, however, is the lack of any mention of the presence of evidences of syphilis. Usually the absence of such evidences is particularly mentioned.

Turnbull [13] discusses the etiology of forty-eight cerebral aneurysms and finds that none of them seemed to be of syphilitic origin, although he recognizes that this is the dominant cause of aneurysm of the aorta, and occasionally in other arteries. In this series he attributed fifteen aneurysms to infected emboli and thirty-three to medial degeneration

9. Virchows Arch. f. path. Anat. **65**:385, 1875.

10. Wichern: Deutsch. Ztschr. f. Nervenh. **44**:220, 1912.

11. Weidman, Fred D.: Ruptured Aneurysm of the Right Vertebral Artery, J. A. M. A. **65**:1105 (Sept. 25) 1915; Am. J. M. Sc. **151**:427, 1916.

12. Compt. end. Soc. de Biol. **69**:210, 1910.

13. Turnbull: Brain **41**:50, 1918.

following congenital developmental deficiency. Hedinger[14] reports an aneurysm (1 cm. by 4 to 5 mm.) in the left vertebral artery observed shortly after a fall backward downstairs with injury to the neck and shoulders, and believes that the aneurysm was of traumatic origin since there were no evidences of arterial disease elsewhere, and no signs of syphilis. He speaks of having had also three cases of ruptured aneurysm of the "arteria vertebralis et basilaris" in syphilitic patients, without giving details.

Several other writers on intracranial aneurysm have attributed importance to traumatism in their etiology. It is indeed easy to imagine that the larger arteries may readily suffer traumatism from violent blows on the skull, since we know from the evidence concerning fractures and cerebral contusions *a contre-coup*, that sudden alterations in intracranial relations take place that must almost certainly put a sudden severe strain on the large vessels at the base on account of their fixation to the skull.

Direct traumatic aneurysms in the cervical portion of the vertebral artery have been described, especially from wounds received in the World War, but these do not concern us here. The literature has been largely collected by Kuttner[15] and Kausch.[16]

The evident rarity of large intracranial aneurysms in the vertebral arteries makes it desirable to put on record a case that I have recently examined, despite the fact that no adequate neurologic examination was made before death. This is especially regrettable since the size and location of the aneurysm were such that symptoms of a cerebellopontile angle tumor should certainly have been present, and we know that some at least were.

REPORT OF A CASE

The subject was a colored man, 54 years old, who entered the Illinois Central Hospital on Dec. 21, 1920, complaining of pain in the left side, loss of weight and weakness. He had difficulty in urination; the urine was scanty, and the patient had had nocturia during the last three months, getting up two or three times during the night and passing only a small amount of urine. He had had some dizziness since September, during which month it was marked. He had also diminished vision.

The patient gave a history of typhoid, pneumonia and measles during childhood, and malaria twenty-two years ago. He used whisky slightly and admitted having had gonorrhea but no chancre. His wife was living and well; she had not had children, but had an abortion soon after marriage. Two of his brothers were dead, and he had four sisters who were living and well. The man had been ill for about four months; he became ill in bed in September and had worked two weeks since that time.

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14. Hedinger: Cor.-Bl. f. schweiz. Aerzte **47**:1393, 1917.
15. Kuttner: Beitr. klin. Chir. **108**:1, 1917.
16. Zentralbl. f. Chir. **45**:885, 1918.

Physical Examination (made in the hospital).—The pupils reacted to light and accommodation and were regular. The patient expectorated large quantities of whitish yellow sputum having a foul odor. The lungs showed dulness over both bases. Mucous râles were present over both sides anteriorly. Several teeth were missing and one was carious. The tongue was furred. The tonsils were hidden. No paralyses of any kind were noted.

The apex beat was under the nipple at the fourth interspace. The left border of the heart was 2 cm. outside the nipple. The right side was under the sternum. The heart sounds were normal.

The abdomen was flat, contained no fluid, showed no tenderness and no organs were palpable. The genitals were normal. There was a slight inguinal adenopathy. The knee reflex was absent, the patient being moribund.

Roentgen-ray examination showed pyorrhea of all the remaining teeth. The heart was greatly enlarged, lying in a transverse position, greatly distended and pulsating heavily. The urine showed a few pus cells and hyalin casts. While in the hospital the patient coughed a great deal, especially when trying to swallow liquids, which he swallowed with much difficulty.

Course of Illness.—On January 1, the patient fell out of bed. On January 2, he was unable to take food and coughed a great deal; his breathing was labored; he still swallowed with difficulty. On January 4 he was semiconscious, the pulse and the general condition were poor.

The temperature fluctuated between 101 and 103 F. until the last day when it rose from 99 to 106. The pulse increased from 100 on the day of admission to 160 on the day of death, January 5.

These findings merely indicated a bilateral pneumonia, probably hypostatic, and in the advanced stage of this terminal infection, with the patient moribund most of the time, no evidence of nerve lesions was detected. Evidently there were no marked facial or ocular paralyses, since doubtless these would have been observed. After the necropsy the widow was interviewed, and we obtained the following additional facts:

History Obtained from Widow.—When the patient was about 18 years old, while walking on a railroad trestle, he was struck by a train. He was knocked to the ground about 12 or 15 feet (365.76 to 457.2 cm.) below, and his forehead hit a rock. He remained unconscious for three or four days. The injury on his forehead was described as being about "the size of a fifty-cent piece." He was treated at home and no surgical procedures were employed. As far as could be ascertained, there was no immediate functional effect, but the patient had said that he had felt foolish for some time afterward. He was married when 23. No history was obtainable of his condition in the interval between injury and marriage. He was apparently well until eight or ten years ago when he began complaining of headaches. At times he would sit and hold his head in his hands because of the pain. He complained particularly of pain at the top of his head where it felt "as if the flesh was nailed to his head." These headaches became worse. He worked as an elevator man for the last five years before his death.

About three years ago, while sitting in a chair, the patient had what his wife called a "spell." He shook all over for about ten or fifteen minutes, after which he had a high fever and sweated profusely, and said his eyes felt as if they were crossed. A physician was called, who advised him to stay in bed. He remained in bed five days and at home twelve days. His body was tender all over; he could not stand to be rubbed with a liniment. A physician

prescribed mercury for him. He went back to work but was very weak for one week. A few months later in the same year he had another similar attack. During the attack his mind was clear. He went to work the next morning but was weak. Two months later he had a third attack and went to work the next morning but was weak the day following.

The following autumn he complained of his teeth and was advised to have them extracted. About three months later two teeth were extracted. A few months afterward he came home sick, still complaining of his teeth. He was advised by the company physician to have more teeth extracted, which was done, but no relief was obtained. He felt as if he were cross-eyed. He was fitted with two pairs of eyeglasses but obtained no relief. He was always complaining of roaring in his ears. After having the teeth extracted he went to work. One day in November, 1920, while at work, he had what was called a paralytic stroke. He fell down in the elevator and had to be assisted to his feet. Information was not obtained as to whether he became unconscious or not. He recovered from this attack and came home alone, but later found he could not walk steadily. About three weeks later he complained that his legs felt like lead although he was able to move them slightly. He lost control of urination and stools, became unable to articulate and could not swallow. He grew steadily worse. His eyes were affected so that he could not recognize objects held before them. Apparently no facial paralysis existed. He was then taken to the hospital.

The records of the company physician contain no reference to any symptoms that could be referred to the intracranial tumor, and physicians who saw him daily at his work observed no evidence of any facial paralysis.

Necropsy Examination.—This examination was performed about eighteen hours after death.

External Appearance: The body was that of a well nourished and well developed colored man about 54 years old. There was no icterus. The superficial lymph glands were not noticeably enlarged. There was no edema of the ankles. The edges of the tibiae were smooth and there were no scars on the external genitals. There was about 1 cm. of subcutaneous fat. A small fatty hernia of the round ligament of the liver was found in the midline, about 2 cm. in diameter.

Pleural Cavities: The left pleural cavity was free from fluid and from adhesions except between the pericardium and the base of the left upper lobe. The thymus was replaced by fat.

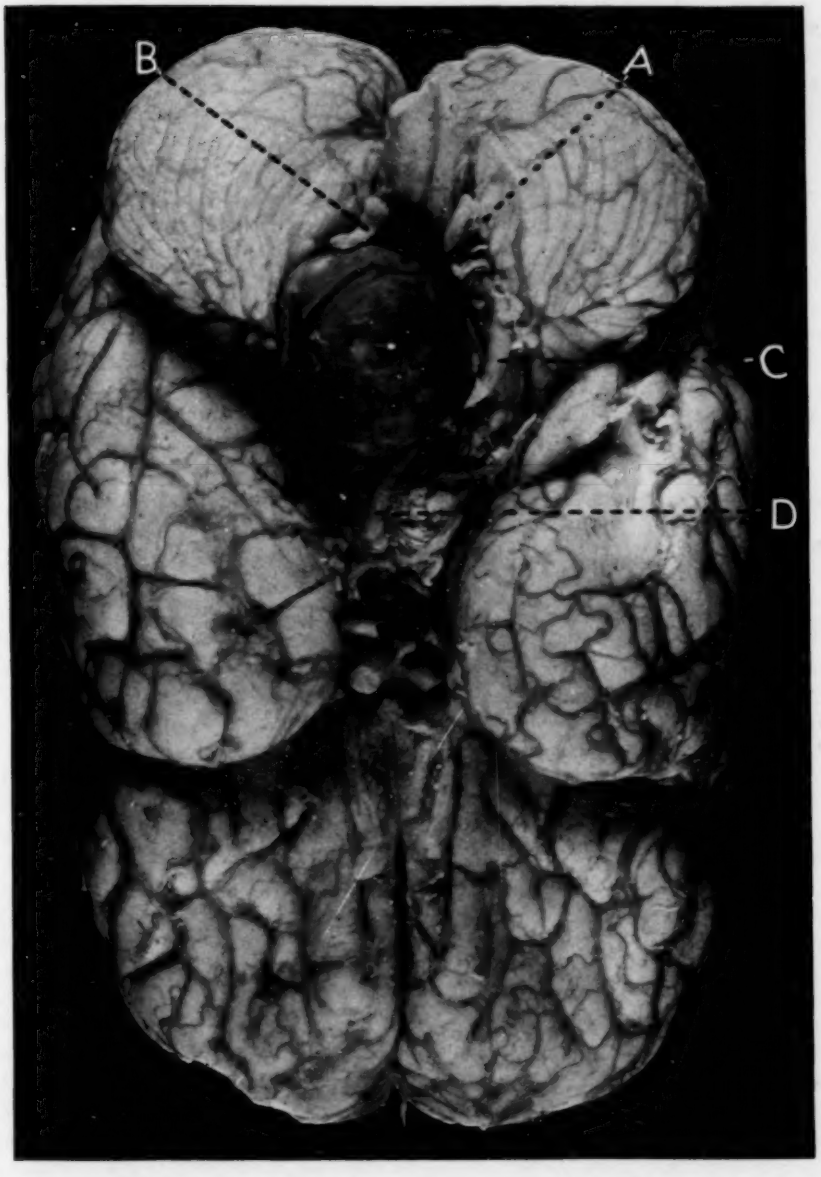
The right pleural cavity contained a few adhesions along the anterior margin of the lower lobe. Elsewhere it was free from adhesions and contained no fluids.

Pericardial Cavity: This was normal.

Mouth and Pharynx: Many teeth were missing, and there was infection of the gums around those remaining. Otherwise there were no changes.

Larynx and Trachea: These showed no changes.

Heart, Aorta and Vessels: The heart was considerably enlarged, chiefly by hypertrophy of the left ventricle. The coronary vessels from the outside showed many calcified spots. The inner surface of the coronary arteries showed numerous yellow sclerotic patches. The valves were normal. There were extensive subendocardial hemorrhages in the left ventricle, chiefly in the upper portion. The myocardium was apparently normal except for a few small areas of fibrosis. The wall of the left ventricle was 15 mm., of the right ventricle



A, aneurysm of the left vertebral artery. Note that it has crowded and compressed the medulla upward and to the right. The defect observed was produced by removing material for sections. *B*, left vertebral artery which abruptly enters the aneurysmal sac; *C*, right vertebral artery; very sclerotic and crowded to the right; *D*, basilar artery.

3 to 4 mm., thick. There were a few small sclerotic patches in the arch and descending portion of the aorta. Both cavities of the heart were enlarged. The estimated weight of the organ was 400 gm.

Lungs: These showed marked bilateral hypostatic congestion with bronchopneumonia. The peribronchial lymph glands were extremely anthracotic but showed no evidences of caseation or calcification.

Liver and Gallbladder: These organs were normal.

Spleen: The spleen was small and distinctly fibrotic but not hard. There were fibrous adhesions between the spleen and the stomach.

Pancreas: The pancreas was small but normal in appearance.

Gastro-Intestinal Tract: The stomach showed numerous submucous hemorrhages, especially in the cardia, but was otherwise normal. The intestines showed no abnormality.

Suprarenals: The suprarenals were normal.

Kidneys: The kidneys were slightly smaller than usual. The cut surface of the left kidney showed the cortical markings well preserved. The capsule stripped readily leaving a smooth surface. The right kidney was also normal.

Urinary Tract: The urinary bladder was normal.

The prostate was somewhat enlarged. Nodules of hyperplasia were present in the median lobe, which protruded slightly under the mucosa of the bladder.

The seminal vesicles were normal.

There were numerous calcified thrombi in the periprostatic veins.

Lymph Glands: In general, these were normal.

Brain and Meninges: There was some rachitic deformity of the skull. The dura was very thin. The veins on the surface were distended. The vertex of the brain was in close contact with the dura. There was a small amount of clear cerebrospinal fluid. The sulci of the brain were much flattened, and the lateral ventricles were greatly dilated, each measuring 25 mm. across in the widest parts. The hypophysis was normal. There was a nearly spherical, chocolate-brown tumor lying exactly at the junction of the pons and the medulla, both of which were compressed (see illustration). The tumor was nowhere adherent but was held in place by the blood vessels which entered it. About 80 per cent. of the tumor lay to the left of the median line. The basilar artery was crowded to the right and the left vertebral artery entered the tumor at a point near the inferior external angle. This artery reappeared again at a point 2 cm. nearer the midline at the same level, at a point 7 mm. from its junction with the basilar artery. The covering of the mass was continuous with that of the artery. The mass measured 34 mm. in its anteroposterior diameter, 30 mm. transversely, and 35 mm. vertically. The superior surface forced itself into the space formed by crowding the medulla from the cerebellum, reaching a point about 15 mm. above the inferior surface of the medulla. It was filled with a mass of dark, granular clot, somewhat laminated, with numerous calcified areas near the arterial side, and no fluid blood. The shape of the aneurysm had been partly molded by the adjacent brain tissue, for there was a somewhat pyramidal projection of the superior surface into the angle between the cerebellum, medulla and pons. The basilar and vertebral arteries were very sclerotic with areas of calcification, and the other cerebral arteries also were sclerotic.

The left vertebral artery had an extremely thick wall in the portion proximal to the aneurysm, with only a minute lumen remaining. Distal to the aneurysm it was occluded by a thrombus up to the point where it entered the basilar

artery. The left vertebral artery was only about one half the size of the right, which was of the same diameter as the basilar artery. Apparently the left vertebral artery had been occluded for a long enough time to permit secondary sclerosis of the left and compensatory dilatation of the right vertebral artery. There were a few soft protrusions from the surface of the tumor which was covered by a smooth capsule. The cerebellum was slightly compressed at the anteromesial angle, and the medulla was pushed upward and to the right, being distinctly curved. The sixth to ninth left cranial nerves were flattened between the aneurysm and the cerebellum. The anterior margin of the tumor was just posterior to the fifth nerve origin.

There was a distinct grooving at the base of the skull involving the sphenoid bone and the occipital bone at the site where the tumor lay, just anterior to the foramen magnum. The dura was intact at this place, and the tumor was not adherent to it, nor was the dura abnormally adherent to the skull at this point. No evidence of any old injury to the skull was observed.

Microscopic examination of the wall of the aneurysm showed it to be composed solely of a thin membrane of dense hyalinized fibrous tissue, in which no elastic tissue or other remains of the artery wall could be detected, at least in the portion examined. The sac contained masses of clotted blood, much disintegrated, with no evidence of infection. There were a few small calcified masses attached to the vessel wall but within the clot. There was no evidence of organization of the clot, and no inflammatory reaction in the wall of the aneurysm.

The aorta showed merely a simple senile sclerosis. Neither here nor elsewhere could evidence of syphilis be found. No microscopic changes of importance were found in the other tissues.

Anatomic Diagnosis.—The anatomic diagnosis was: Saccular aneurysm of the left vertebral artery, with compression of the pons, cerebellum and medulla, and roots of the sixth to the ninth cranial nerves; pressure deformity of base of the skull; dilatation of the cerebral ventricles; bilateral hypostatic bronchopneumonia; diffuse senile arteriosclerosis; hypertrophy of the left cardiac ventricle; slight fibrous myocarditis; ventral fatty hernia; slight rachitic deformity of the skull and tibiae; subendocardial hemorrhages.

SUMMARY

A colored man, with no clinical or anatomic evidence of syphilis, but with a history of severe cranial traumatism many years before, died of hypostatic bronchopneumonia, without giving marked evidence of increased intracranial pressure or of cranial nerve paralysis during his last illness. Necropsy examination revealed a large aneurysm of the left vertebral artery, causing much deformity about the cerebellopontile angle, compressing the sixth to ninth cranial nerves, occluding the left vertebral artery, and, by pressure on the aqueduct of Sylvius, causing distinct internal hydrocephalus. That the swelling had been present for a long time was indicated by the presence of a distinct grooving in the floor of the skull beneath it. A necropsy inquiry revealed that the patient had had roaring in the ears, dizziness and "spells" three years before his death. Neuralgic pain and infection about the gums led

to the extraction of several teeth four months before death. Two months later there was an attack of syncope. Three weeks before death difficulty of speech, decreased vision and difficulty in swallowing appeared, terminating in hypostatic bronchopneumonia.

As far as can be learned from the literature, this seems to be one of the largest vertebral aneurysms yet observed. It is unfortunate that the patient had not been the subject of a thorough neurologic examination during life, for the differentiation from a cerebellopontile angle tumor would have afforded difficulty. In two or three recorded cases a bruit has been heard over a vertebral aneurysm, but the clue would not have been present here since the artery was completely occluded. This aneurysm corresponds to the statement in the literature that intracranial aneurysms are not usually the result of syphilitic arteriosclerosis, but that they often give a history of antecedent cranial trauma. The presence of a distinct grooving of the skull beneath the aneurysm, without adhesion or erosion, would support the hypothesis that the aneurysm might have formed after the injury to the 18-year-old boy. Probably this yielding of the skull permitted the aneurysm to reach the exceptionally large size it had attained.

A SILVER DIFFUSION METHOD FOR STAINING NERVE FIBERS IN PARAFFIN SECTIONS*

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The method detailed below varies little from that described by Warthin and Starry[1] for the demonstration of spirochetes in paraffin sections. The only modification has been the substitution of a gelatin film for the capillary space between the two coverslips. Warthin and Starry attached paraffin sections to coverslips, washed them in silver nitrate solution, pressed a second coverslip over the section, and allowed silver nitrate solution to diffuse in between the two coverslips.

When their method was used on a suspected gumma of the brain, it was found that numerous fibers were stained, but none of them could be identified as spirochetes. With the idea of determining the possibilities of the stain in regard to nerve fibers, an investigation was undertaken which resulted in obtaining such preparations as are shown in the illustrations.

One difficulty with the Warthin-Starry coverslip method was the uneven character of the silver deposit. This was done away with by embedding the coverslip, face up, in a warm 10 per cent. gelatin solution, allowing it to harden, and then pouring silver nitrate on the surface. The silver nitrate then diffused down to the section. Later the gelatin was melted off and the coverslip immediately immersed in the developing solution exactly as recommended by the authors cited. Even staining, so much desired in all the silver methods was usually obtained.

The method has its limitations. It is not adapted to anatomic work since it does not stain the finest ramifications of the nerve fibers, nor such fibers as occur in the deeper layers of the retina. Due to shrinkage, the finest pericellular networks cannot be brought out in any but perfect preparations. It has advantages over the Bielschowsky frozen section method in that larger sections are used, even the whole cross section of the pons; the tissues are cut thinner, and the relations are maintained. The neurofibrillae are well shown when not altered by disease. Another advantage is that twin preparations can be made with consecutive sections, the one showing the cell processes and neurofibrilae outlined in silver, and the other showing the identical cells stained by Nissl's method to show the tigroid bodies.

*From the Laboratory of Neuropathology, Philadelphia General Hospital.

1. Warthin, A. S., and Starry, A. C.: Second Improved Method for the Demonstration of Spirochaeta Pallida in the Tissues, J. A. M. A. **76**:234 (Jan. 22) 1921.

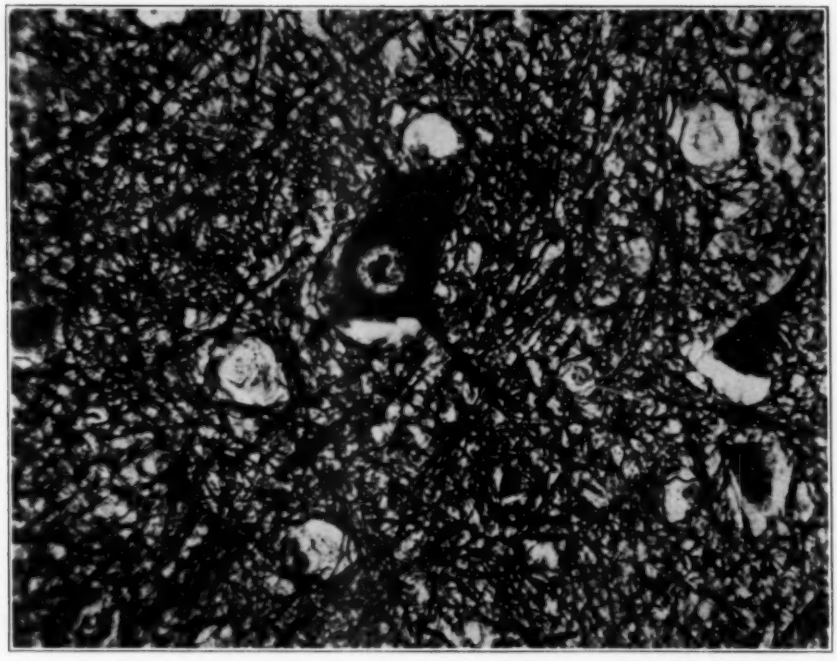

Fig. 1.—Precentral cortex showing Betz cell stained for nerve fibers by silver agar coverslip method.

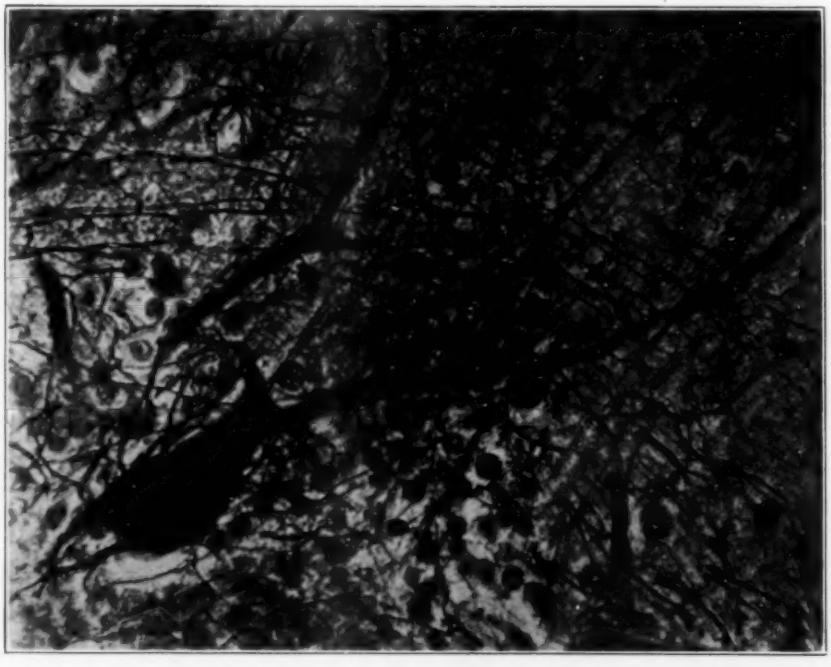

Fig. 2.—Purkinje cell showing basket fibers.

Fig. 3.—Multiple sclerosis; longitudinal section of sclerotic patch stained for axis cylinders.

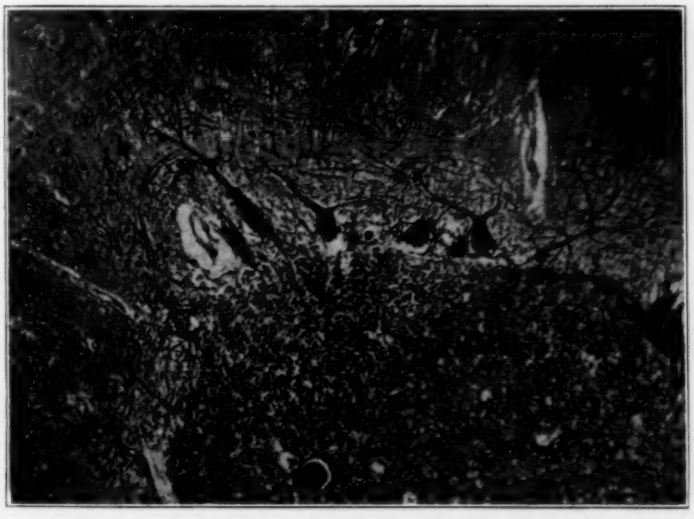

Fig. 4.—Cerebellum; silver diffusion preparation to show nerve cells and processes.

TECHNIC

1. Formaldehyd of alcohol fixation. (Alcohol fixation permits toluidin blue to be used for cell stain with brilliant results.)

2. Paraffin embedding. Cut sections from 5 to 8 microns. These are attached to coverslips which have previously been coated with albumin fixative and dried in the oven over night. They are then blotted and placed in the paraffin oven for from fifteen to thirty minutes.

3. The paraffin is then removed by xylol and the coverslip carried through graded alcohols to water.

4. The coverslip is placed, section side up, in a staining dish the bottom of which is covered by a 5 mm. layer of melted 10 per cent. gelatin, freshly made. The gelatin is allowed to harden and 10 c.c. of 2 per cent. silver nitrate is poured on the surface. This is kept in the dark for twenty-four hours or more. The depth of the gelatin may be increased in order to obtain detail, a longer time being given for diffusion.

5. The gelatin is removed by holding the dish, bottom up, under a hot water tap until the gelatin falls away.

6. The coverslip is plunged without washing into the developer and kept moving until the section is deep brown.

7. It is then washed in water, fixed in 5 per cent. sodium hyposulphite solution, washed again, carried through graded alcohols to xylol and mounted in balsam.

The developer is made as follows:

Glycerin Gelatin (10 per cent.) Agar agar (1.5 per cent.)	warm, of each	5.0 c.c.
Silver nitrate (2 per cent.)		3.0 c.c.
Hydroquinone (5 per cent.)		0.7 c.c.

The glycerin, gelatin and agar agar are added in turn to the silver nitrate, and then the pyrogallol stirred in rapidly. The section is immediately immersed.

All solutions should be fresh and should be made with distilled water. Metal forceps should be coated with paraffin when used in the developer.

THE TREATMENT OF GENERAL PARESIS BY THE INTRACISTERN ROUTE *

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To be conservative in the adoption of new procedures is advisable. This is especially true of the clinical application of puncture of the cisterna magna for diagnostic and therapeutic purposes, owing to the proximity of the medulla oblongata. Wegeforth, Ayer and Essick [1] in their pioneer work have demonstrated the practical value of puncture of the cisterna cerebellomedullaris and the ease with which it is done, after careful anatomic studies on the cadaver. Ayer [2] reports forty-three punctures and says cistern puncture is indicated in the following conditions: (1) spinal subarachnoid block following meningitis; (2) combined with lumbar puncture for irrigation of the subarachnoid space; (3) as a route for injection in epidemic meningitis; (4) in conjunction with lumbar puncture in diagnosis of cord compression.

The object of this paper is to discuss puncture of the cisterna magna in the treatment of general paresis. A total number of 250 punctures has been made in twenty-eight patients.[3] No accidents resulted from the punctures. At the present time I do not feel justified in drawing any conclusions as to results.

Intracistern therapy in general paresis appears to have several advantages. The facility with which cisternal puncture can be performed is a great advantage over the intracerebral method. Treatment is doubtless more intensive than by the intraspinal method since the injection is made nearer the seat of the disease and results in less dilution with probably more widespread dissemination of the serum. Ink injected in the lumbar subdural space, in the same dosage as the serum employed in treatment, has shown little staining at the base of the brain. Intracistern injection of arsphenamized serum, in proper hands, is a safe procedure. There is evidence that this method is rendered more effective following intravenous administration of hypertonic salt solution, for Weed and McKibben [4] in their papers

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* From the New Jersey State Hospital at Trenton.

1. Wegeforth, Ayer and Essick: The Method of Obtaining Cerebrospinal Fluid by Puncture of the Cisterna Magna, Am. J. M. Sc. **157**:789 (June) 1919.

2. Ayer, J. B.: Puncture of the Cisterna Magna, Arch. Neurol. & Psychiat. **4**:524 (Nov.) 1920.

3. Since this paper was presented for publication the total number of punctures has reached 550 in forty-five patients. There were no noteworthy changes in the clinical and serologic results.

4. Weed and McKibben: Pressure Changes in the Cerebrospinal Fluid Following Intravenous Injection of Solutions of Various Concentrations, Am. J. Physiol. **48**:512 (May) 1919. Experimental Alteration of Brain Bulk, Ibid., p. 531.

have demonstrated that this is followed by a marked fall in cerebrospinal fluid pressure. It seems reasonable to suppose that there will be deeper penetration if serum is administered following this fall in pressure. In this way it may be possible to reach all areas of the brain and syphilitic foci whether the infection is parenchymatous, endarteritic or meningitic. Hypertonic salt solutions have been used in several cases and in each case a marked fall in cerebrospinal pressure has been noted.

At present there seems to be no unanimity as to the best method of injecting arsphenamized serum in the treatment of neurosyphilis. This applies to intraventricular as well as to intraspinal injections and to spinal drainage with intensive intravenous injections. Intracistern injections of serum would seem to be indicated in preference to the intraspinal method since it has been shown by Dandy, in his experimental studies of hydrocephalus, that from three-quarters to four-fifths of the cerebrospinal fluid is absorbed from the subarachnoid spaces of the brain while only one-quarter to one-fifth is taken up from the spinal subarachnoid space. The cisterna magna is the most important distributing reservoir for the other cisternae, and from these the course is to the subarachnoid space and sulci of the brain where the curative serum is probably absorbed. As before stated, I think that this absorption is probably accelerated after the intravenous injection of hypertonic salt solution. In syphilitic infections of the meningitic type, I feel that intracistern therapy is clearly indicated and should give striking results, although I have not had such a case.

Few attempts have been made to treat neurosyphilis by the intracistern method. Berriel,[5] at the University of Paris, has injected arsphenamized serum through the sphenoidal fissure into the large basal subarachnoid cistern. In this country this method has been employed by Kopetzky and Haynes,[6] who have tried it with success in the treatment of meningitis. With the exception of Ayer's work, little has been done with the cisterna magna approach. McKusker,[7] working at the Butler Hospital, published his initial case in October, 1920. Since that time he has been using this method.

METHOD OF TECHNIC

The technic has been fully described by Wegeforth, Ayer and Essick [1] and more recently by Ayer.[2] There is little that can be added to these careful descriptions. The patient assumes the posture (on

5. Berriel, M. L.: Bull. et mém. hôp. de Paris **35**:986, 1914.

6. Kopetzky, S. J., and Haynes, I. S.: Meningitis, Cornell Univ. Monograph, 1912.

7. McKusker, H.: Some Observations on Cistern Puncture, J. Nerv. & Ment. Dis. **53**:453 (June) 1921.

his side) for lumbar puncture with the neck moderately flexed—facilitated by a pillow under the head. Great care is taken to secure careful alignment of the vertebral column. The skin is sterilized (iodin-alcohol) after careful shaving and cleansing. After the first ten punctures no local anesthesia is employed although cocain is used if the patient desires it. The spine of the axis is carefully located by the index finger. The standard 18 gage nickeloid lumbar puncture needle is used. After perforating the skin just above the axis the needle is pushed upward and forward (anatomically) directly toward the glabella. The external auditory meatus is in the plane of this line and is a handy landmark in determining the inclination of the needle. The dura is pierced at a depth of from 3 cm. to 5 cm. Following Ayer's method, a deadline consisting of a faint circular scratch is made 6 cm. from the tip of the needle.[8] The greater the angle or the nearer one comes to the posterior margin of the foramen magnum, the greater the margin of safety since the cistern is of greatest depth at this point.

No clinical punctures were made until careful work on the cadaver had made essential landmarks and relations clear. On the cadaver cistern puncture is much easier to perform than puncture of the lumbar subarachnoid space. Pathologists could advantageously use this method for obtaining fluid for cultivation and examination and for fixation. The interspaces are approximately twice as wide as that between the third and fourth lumbar vertebrae. The sensation on piercing the dura is more definite than that of the lumbar space, and practice enables one to tell when the cistern is entered. Frequently, however, it is necessary to withdraw the stylet before completing the puncture. After one has succeeded in getting rid of the initial apprehension, the technic is easy. It is worthy of note that of our total (250) only six punctures were repeated; two of these were due to movements and poor cooperation by the patient and the others to failure of the operator to locate the spine of the axis and consequently attempting to puncture outside the midline. Attempt at puncture in a negro was given up when no fluid was obtained at a depth of 6 cm. Later it was repeated and owing to the thickness of the skin no fluid was obtained until a depth of 6.5 cm. had been reached. Bloody fluid is less frequently encountered than in lumbar puncture.

We employed the usual Swift-Ellis technic for the preparation of the arsphenamized serum. Our patients were subject to the usual varying ward conditions, and treatment in many cases was at irregular intervals. On Monday of each week the patients were given intravenous injections of 0.3 of a gram of arsphenamin by the usual gravity or syringe method. After the serum was prepared by the standard

8. A thread tied about the needle serves as well.

technic, it was reinforced by the addition of from 0.5 to 1 mg. of arsphenamin. Serums were diluted to the average of 25 c.c. Intracistern injections were given on Tuesday, slowly by the gravity method after drainage of the same amount of fluid. In several cases we drained from 60 to 80 c.c. with no ill effects. In three of our patients we gave hypertonic solutions preceding intracistern injections. In each case we injected intravenously 50 c.c. of the 30 per cent. sodium chlorid solution, just after the cistern had been punctured. We were fortunate in having good cooperation and were able to record pressure changes for periods varying from 15 to 30 minutes, by attaching a manometer to the needle. After the injections of hypertonic solution the fall in pressure was found to correspond to that which has been noted by many observers. Ebaugh and Stevenson[9] have shown the clinical application of this procedure by demonstrating pressure changes in a case of epilepsy. The accompanying curves show the results of these experiments in two cases. It is interesting to note that the change in pressure in each case occurs immediately after intravenous injection is begun.

RESULTS OF PUNCTURE

We wish to note our observations on: (1) subjective changes during puncture; (2) complaints after puncture; (3) clinical results and serum changes.

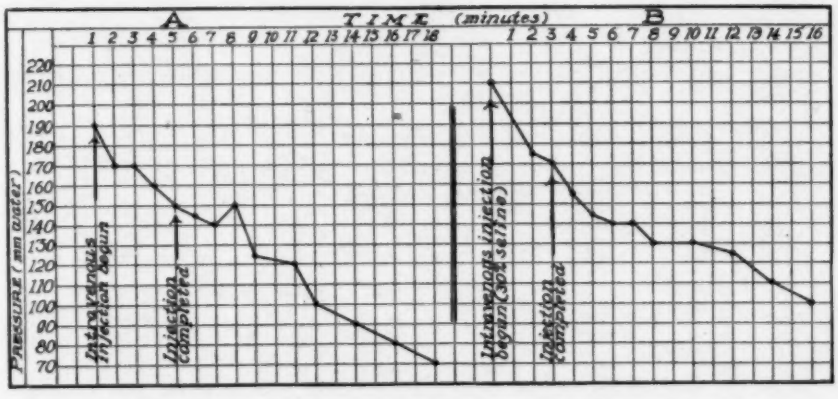
These cases represent the average state hospital types of the disease. Unfortunately all of our cases were of long duration. From the psychiatric point of view the diagnosis was definite. They showed, briefly, the following changes: profound sensorium alteration consisting of various degrees of mood alteration and disorientation; marked discrepancies of memory, retention, grasp of general information and calculation; poor judgment, typical speech and writing defects. Neurologically they presented the usual pupillary and reflex changes, facial tremor and in many cases the findings of focal types of this disease. Serologically they showed a typical paretic colloidal gold reaction with increase in globulin and pleocytosis with a positive blood and spinal fluid Wassermann reaction. An attempt has been made to consider the clinical findings and changes separately from the serologic ones.

Changes During Puncture.—The first ten punctures were made after cocainizing the skin. Few difficulties were encountered, although occasionally the needle had to be depressed on touching the edge of the occipital bone. The patients rarely complained of pain and several of

9. Ebaugh, F. G., and Stevenson, G. S.: Measurements of Intracranial Pressure Changes in an Epileptic and Its Experimental Variations, Johns Hopkins Hosp. Bull. **31**:440 (Dec.) 1920.

them spontaneously asked for the "neck punctures" in preference to the "back punctures." After insertion of the needle the patients did not complain. There were no changes in pulse, respiration or blood pressure, and no vasomotor changes. Cerebrospinal fluid pressures were taken in each case. These demonstrated that the same pressure relations existed in the cisterna as in the lumbar subarachnoid space. The average pressure was about 160 mm. of water; the highest recorded was 325 mm., the lowest 100. Generally from 20 to 30 c.c. of fluid were withdrawn and immediately replaced with serum which was allowed to run in slowly by the gravity method. With one exception, the patients never complained of pain while this was being done.

Changes After Puncture.—Following the injection the patients remained on the table for several minutes. As is usual in cases of



Curves showing the fall in pressure of the cerebrospinal fluid following the intravenous injection of 50 c.c. of 30 per cent. salt solution. *A*, showing the fall in cerebrospinal fluid pressure in patient A. C. from 190 mm. of water to 70 mm. in the course of eighteen minutes. The cisterna magna was punctured after intravenous injection was begun. *B*, showing the fall in cerebrospinal pressure in patient J. N. from 210 mm. of water to 100 mm. in the course of fifteen minutes.

general paresis, the patients rarely complained of headache. They got off the table unassisted and walked to the wards. There were no changes in pulse, respiratory rate or blood pressure, and no vasomotor changes. In no case did nausea and vomiting occur. Two patients complained of headache in the beginning of the treatment, but this was relieved by increasing the periods of rest following puncture. Although this factor is unimportant, it must be studied further. The same reaction frequently occurs with intraspinal injections of arsphenamized serum.

Serologic and Clinical Results.—Our clinical and serologic results thus far have not been striking. Despite the fact that several patients

had received previous treatment, the Wassermann reaction of the blood and spinal fluid in most cases remained unchanged. The cell count and globulin gradually reached normal limits after several intracistern injections of arsphenamized serum. At present twelve patients have shown successive negative globulin reactions. None of them shows pleocytosis. The colloidal gold reaction was slow in changing but tends to follow the globulin decrease. The table gives the present status of the patients treated. In order to prevent confusion we have not charted the serologic findings in detail but characterize the changes as good, bad or slight.

The clinical results of intracistern therapy have been equally disappointing. However, one feels justified in further employment of the method. The clinical results do not check with the serologic findings, as is shown briefly in the table. No noteworthy changes were noted in the mental status of any patient with the exception of the patient in Case 27. This case was of short duration, onset followed trauma and the patient has shown proressive improvement.

CLINICAL AND SEROLOGIC RESULTS TO JULY 1

No.	Name	Intracistern Injections	Serologic Result	Clinical Result	Remarks
1	L. E.	8	Poor	Poor	Previous intraventricular therapy; blood negative
2	J. J.	11	Poor	Poor	Previous intraventricular therapy
3	W. R.	19	Poor	Poor	Previous intraventricular therapy
4	R. D.	6	Good	Poor	Previous treatment
5	M. Z.	11	Poor	Slight	Globulin now negative; cells 7; colloidal gold test negative
6	A. P.	17	Good	Poor	Negative blood and fluid Wassermann reaction; negative globulin and colloidal gold reactions
7	G. G.	5	Poor	Poor	Gradual deterioration
8	L. P.	14	Good	Bad	Serology negative; no clinical improvement
9	E. T.	9	Poor	Poor	No definite clinical change
10	A. L.	14	Good	Bad	Spinal fluid positive, otherwise serology negative
11	A. C.	9	Slight	Bad	Treatment discontinued; gradual deterioration
12	S. M.	10	Poor	Poor	Patient died of pneumonia
13	J. N.	7	Poor	Poor	No noteworthy clinical changes
14	H. S.	8	Slight	Slight	Globulin negative; cells 5; slight change in Wassermann reactions
15	H. B.	7	Poor	Poor	No changes
16	N. S.	5	Poor	Poor	Patient resistive; rapid deterioration; treatment discontinued
17	J. C.	6	Poor	Poor	No improvement
18	F. T.	6	Slight	Slight	Patient discharged; beginning remission?
19	T. H.	4	Poor	Poor	No improvement
20	R. J.	17	Slight	Poor	No definite clinical changes noted
21	S. S.	19	Good	Poor	Serology reported negative; little change in mental status
22	M. F.	5	Poor	Poor	Gradual deterioration
23	W. O.	6	Poor	Poor	No improvement
24	A. H.	7	Poor	Slight	Endarteritic type; slight improvement
25	V. H.	5	Poor	Slight	Slight clinical improvement; patient bedridden on admission; now up; speech defect less marked
26	W. R.	5	Poor	Poor	No change
27	W. R.	6	Good	Good	Marked clinical improvement
28	A. O.	5	Poor	Poor	No changes

SUMMARY AND CONCLUSIONS

Intracistern injections of arsphenamized serum in the treatment of general paresis theoretically has some advantages over other methods. The facility of the operation makes it more practicable than intracerebral injections. The cisterna magna as a distributing center should promote a more widespread dissemination of serum. Complaints of the patients furnish no contra-indication. No reactions occurred. The operation is a safe clinical procedure.

We fully agree with Ayer's landmarks and technic and his dictum that one should not perform cisternal puncture without preliminary experience on the cadaver.

Cerebrospinal fluid pressure reductions were obtained following the intravenous injection of hypertonic salt solutions similar to those first described by Weed and McKibben.

The clinical and serologic results in the cases treated seem to justify further use of this method.

Philadelphia General Hospital.

THE INCIDENCE AND CHARACTERISTICS OF DYSTHYROIDISM AS AN EX-SERVICE DISABILITY

WITH ESPECIAL REFERENCE TO THE FINDINGS IN DISCHARGED SOLDIERS AND SAILORS, EXAMINED FOR THE BUREAU OF WAR RISK INSURANCE (NOW THE VETERANS' BUREAU), DISTRICT NO. 2 *

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The literature of the late World War early recorded a remarkable frequency of hyperthyroid conditions as compared with their incidence in times of peace. This was noted among combatants and noncombatants in military service, and in the civil population as well. Disabled ex-service men and women who had been invalided, and those less severely disabled in service who had been discharged in good condition but who had had a relapse, have together constituted the examinees of the federal Bureau of War Risk Insurance (the Veterans' Bureau since Aug. 9, 1921); here also the incidence of hyperthyroidism has been above normal. H. W. Wright,[1] in a review of postbellum neuroses, separates a group of "secondary neurasthenic states," including those complicated by thyrotoxicoses, or those in which some disorder of the thyroid is a prominent feature. It seemed worth while to review the war literature on dysthyroidisms in the light of postwar conditions—using the term "dysthyroidism" broadly, to include all forms of thyroid anomaly.

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* Permission to publish these data has been granted by Dr. T. M. Baird, medical officer in charge, New York City Station, U. S. Veterans' Bureau, District No. 2. We also wish to express our appreciation of the helpful coöperation of Dr. T. J. Vosburgh, chief neuropsychiatrist, and Katherine D. Smith, special assistant.

1. Wright, Harold W.: Postbellum Neuroses; a Clinical Review and Discussion of their Mechanism, Arch. Neurol. & Psychiat. **3**:429 (April) 1920.

For 1916, before the United States entered the war, a representative clinic [2] of New York City [3] reported 0.92 per cent. of ductless gland disorders among all neuropsychiatric cases; in 1918, that percentage rose to 3.01, but dropped in 1919 to 1.75. Among the Bureau of War Risk Insurance claimants examined at this office during the first half of 1921, various ductless gland disorders (thyroid, thymic, pituitary, polyglandular, etc.) have been found in 3.93 per cent. of all neuropsychiatric cases, or thyroid disorders in 3.36 per cent. Patients with a neuropsychosis and a thyroid disorder simultaneously, made up 0.59 per cent. of the total number of cases (medical, surgical, etc.).

It is clear that the civilian rate of dysthyroidism incidence rose during the war and has fallen since. The ex-service rate is still high. Statistics are not yet available for comparing its incidence year by year in this district. Surgeon J. G. Townsend [4] of District No. 4 reported earlier this year that "the estimated total neuropsychiatric disabilities present in ex-service men directly traceable to military service is 76,588, this number being further classified as follows . . . endocrinopathies 4,823 . . . " These figures amount to a percentage of 6.29. Our own ratio (3.93 per cent.) is much lower, though still high. A large part of this discrepancy would be taken up by the excessive incidence of goiter in districts [5] in which the endemic type is more prevalent than here. The remaining difference can be construed as evidence—further evidence will be adduced later—pointing toward a beginning post-war decline in total dysthyroidisms. While "the peak for the neuropsychiatric cases is not expected to be reached until 1926," [4] the relative frequency of hyperthyroidism should be lessening already.

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2. The New York Neurological Institute Reports enumerate their ductless gland cases together and not by subdivisions, but it may reasonably be assumed that the great majority are cases of dysthyroidism. The 1920 Report cannot be used for comparison since the Institute added a special endocrinologic clinic about the end of 1919, which soon attracted a disproportionate number of cases of ductless gland disorders. The Institute figures are those for males only, while the Bureau figures include a few females; the comparison is approximately correct, which it would not be if the Institute figures for both sexes were taken, since all authorities agree that the incidence of ductless gland disorders is, *caeteris paribus,* far greater in females than in males.

3. Seventh Annual Report of the Neurological Institute of New York for the Year Ended November 30, 1916. Tenth Annual Report of the Neurological Institute of New York for the Year Ended November 30, 1919.

4. Townsend, J. G.: Rehabilitation of the Ex-Service Personnel of the World War, Mil. Surgeon **48**:127 (Feb.) 1921.

5. Addis, T., and Kerr, W. J.: The Relative Frequency in Recruits With and Without Thyroid Enlargement of Certain Signs and Symptoms which Occur in Neurocirculatory Asthenia, Arch. Int. Med. **23**:316 (March) 1919.

French reports particularly have called attention to hyperthyroidism immediately referable to various forms of shock. Léniez [6] describes three cases occurring before 1910, in which cerebral concussion was followed in from one to three months by definite exophthalmic goiter. The same writer saw a number of mild cases of exophthalmic goiter, "formes frustes," following injuries of the head and without previous signs; the struma was seldom large, and true exophthalmos was infrequent. R. Blondel [7] comments on the increase of exophthalmic goiter during the war, both in women and in men. G. Étienne and G. Richard [8] conclude, both from their own observations and from those of others, that cases of exophthalmic goiter occur rather often in active military service. L. Bérard [9] found many cases of exophthalmic goiter, both mild and severe, in military men between the ages of 20 and 45; he rarely saw those in civil life. On the German side, H. Klose [10] treats it as an accepted fact that exophthalmic goiter has been precipitated frequently by the war; he also suggests that the chemical action of gases may have had a share in the etiology. Among American authorities, Crile [11] makes the statement that "iodin alone, adrenalin alone, thyroid extract alone, emotion, exertion, or infection alone, each causes a 'Kinetic Drive' with phenomena similar to those of exophthalmic goiter." According to Harlow Brooks,[12] "emotional shock, fright, or mental trauma is too well established in the etiology of Graves' disease to admit of question."

Hyperthyroidism and war inevitably call to mind the many discussions and investigations of "soldier's heart"—"neurocirculatory asthenia," "effort syndrome," "disordered action of the heart." Was it or was it not hyperthyroidism? Without laboratory tests, we have been disinclined to take a definite stand on this matter. Such patients appearing for examination have tended to separate into three groups

6. Léniez: Sur le goitre exophthalmique traumatique, Progrès méd. **31**:215, 1916.

7. Blondel, R.: L'accroissement d'une cause de stérilité féminine depuis la guerre par le développement de la maladie de Basedow, Bull. gén. d. thérap. **170**:816, 1918-1919.

8. Étienne, G., and Richard, G.: Maladie de Basedow et émotions de bombardements, Bull. et mém. Soc. méd. d. hôp. de Paris, Ser. 3 **42**:1196 (Dec. 20), 1918.

9. Bérard, L.: La maladie de Basedow et la guerre, Bull. Acad. de méd., Paris **76**:428 (Nov. 28) 1916.

10. Klose, Heinrich: Der Kriegs-Basedow Pathogenese, Typen, Verlauf und militärärztliche Bewertung, Med. Klin. **14**:1199 (Dec. 8) 1919.

11. Crile, G. W.: A Physical Interpretation of Shock, Exhaustion and Restoration, London, 1921, p. 171.

12. Brooks, Harlow: Hyperthyroidism in the Recruit, Med. Rec. **93**:476 (March 16) 1918.

instead of remaining together, the cases of some being diagnosed by cardiac examiners and the patients ordered proper treatment without being referred to the neurologists, while others are so referred, but may or may not be given a hyperthyroid diagnosis in addition to that of a cardiac dysfunction, according to the prominence of signs of hyperthyroidism. It would be impossible to estimate the proportions of these three groups. Occasionally we have labeled a case "hyperthyroidism," when the patient's service diagnosis was known to have been "irritable heart" (or an equivalent term); but the thyroid and neurocirculatory diagnoses have not been used interchangeably.

Divergent views on the proper classification of this "irritable heart of soldiers" may be illustrated by referring to the studies of Thomas Lewis,[13] T. B. Barringer,[14] T. A. Smith and D. Bovaird,[15] T. Addis and W. J. Kerr,[5] L. M. Warfield and F. M. Smith,[16] F. W. Peabody, C. C. Sturgis, J. T. Wearn, H. D. Clough, and Edna H. Tompkins,[17] in favor of discrimination between hyperthyroidism and irritable heart; and to those of Harlow Brooks,[12] J. H. Carroll[18] and G. Railliet,[19] who prefer to consider the two conditions identical. The wartime work of Peabody and his collaborators was particularly exhaustive, including tests of epinephrin sensitiveness and basal metabolism, and finally led them to the conclusion that hyperthyroidism did not play a significant rôle in the production of the syndrome known as the "irritable heart of soldiers."

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13. Lewis, Thomas: Med. Res. Committee, Special Rept. Ser. No. 8, London, 1917.

14. Barringer, T. B., Jr.: "Disordered Action of the Heart" in British Soldiers, J. A. M. A. **69**:1726 (Nov. 17) 1917.

15. Smith, Thayer A., and Bovaird, David: Irritable Heart or Effort Syndrome, Am. J. M. Sc. **156**:872 (Dec.) 1918.

16. Warfield, L. M., and Smith, F. M.: Studies on Irritable Heart. II. Etiology of Irritable Heart, J. Lab. & Clin. Med. **5**:75, 1919-1920.

17. Peabody, F. W.; Clough, H. D.; Sturgis, C. C.; Wearn, J. T., and Tompkins, E. H.: Effects of the Injection of Epinephrin in Soldiers with "Irritable Heart," J. A. M. A. **71**:1912 (Dec. 7) 1918. Wearn, Joseph T., and Sturgis, Cyrus C.: Studies on Epinephrin. I. Effects of the Injection of Epinephrin in Soldiers with "Irritable Heart," Arch. Int. Med. **24**:247 (Sept.) 1919. Tompkins, Edna H.; Sturgis, Cyrus C., and Wearn, Joseph T.: Studies on Epinephrin. II. The Effect of Epinephrin on the Basal Metabolism in Soldiers with "Irritable Heart," in Hyperthyroidism and in Normal Men, Ibid., p. 269. Peabody, Francis W.; Wearn, Joseph T., and Tompkins, Edna H.: Basal Metabolism in Cases of the "Irritable Heart of Soldiers," Med. Clin. North America (Sept.) 1918.

18. Carroll, John H.: Neurocirculatory Asthenia (Soldiers' Heart), Am. J. M. Sc. **158**:35 (July) 1919.

19. Railliet, G.: Basedowisme post-émotionnel et hypertrophie des parotides, Bull. et mém. Soc. méd. d. hôp. de Paris, Ser. 3 **42**:1151 (Dec. 6) 1918.

AUTHORS' MATERIAL

The period covered by our survey dates from Jan. 1, 1921, to July 1, 1921; the cases are those of all former ex-service men and women in this district that had filed claims for compensation from the government on account of disabilities acquired in connection with their military service, and who were consequently examined by the Bureau of War Risk Insurance at the headquarters of District No. 2 in New York City. We include all persons seen at this central office, whether coming from the city or sent in from greater distances for special examinations, and also include city house calls made from the central office. Cases of incidental complaints among war risk beneficiaries in federal training for rehabilitation (under the then "Federal Board for Vocational Education") are not included.

For this period the daily reports of general initial examinations and general reexaminations show a total of 18,675, exclusive of special examinations and consultations. In determining the number of persons examined, which must be less than the total number of examinations because many men received complete reexaminations within the six months, a review of 500 cases showed that 421 of that number were examined once during the set period, seventy-one twice, seven three times, and one four times. This is at the rate of 1,176 examinations per 1,000 persons. Proportionately, the total of 18,675 general examinations represent 15,880 persons examined. Of these, 2,797 claimants received neuropsychiatric examinations in addition to the general examinations (that is, approximately 3,290 neuropsychiatric examinations were made). Among those 2,797, the neuropsychiatric total, ninety-four examinees were found to have some form of thyroid disorder in addition to their nervous or mental complaint. A few goitrous cases with no subjective or objective neurologic symptomatology may have escaped the neuropsychiatric section, but it has been the policy to require a neuropsychiatric examination in all cases of endocrinopathy.

CLASSIFICATION OF CASES OF THYROID DYSFUNCTION

Cases that make up a series showing thyroid dysfunction can be theoretically separated by symptoms into four general groups: (1) toxic thyroid, (2) dysthyroid (in the narrowest sense), (3) hypothyroid, and (4) simple goiter. Group 2 connotes cases of disturbed thyroid activity in which neither hyper-activity nor hypo-activity can be said to dominate the picture. None of these cases was observed. Group 3 is self-explanatory; similarly, no examples were found. (Very few patients had been operated on, and those rather recently.) Group 4 comprises cases of goiter, whether cystic or adenomatous, in which no evidence of dysfunction was determined but in which there was some concomitant neurologic or psychiatric disorder; only one case could be so classified.

Group 1, comprising ninety-three cases, calls for special attention and subdivision. Various fundamenta divisionis are found in the literature. We have taken the well-recognized clinical types, exophthalmic and toxic nonexophthalmic, with a transitional group of "mixed" cases. Our data do not justify an attempt to classify by the presence or absence of hyperplasia.[20] Similarly the conceptions [10] of struma basedowiana as opposed to struma basedowificata, and those of idiopathic and symptomatic exophthalmic goiter, have been kept in view while not being used primarily. "Sympathicotonic" and "vagotonic," [21] however, are feasible as well as valuable conceptions; while their limits are not strictly coterminous with those of toxic exophthalmic and toxic nonexophthalmic goiter, they are sufficiently so to warrant mutual comparisons among findings classified by the two sets of criteria. The nature of examining work in the Bureau of War Risk Insurance has not permitted intensive study of the symptomatology of any group, nor has there been an opportunity to determine reactions to epinephrin injection, thyroid feeding, basal metabolism or other tests. These have been conducted only in the Public Health Service hospitals, or others to which patients could be referred for intensive study and treatment, and are outside the scope of this paper.

Five exophthalmic, sixty-three toxic nonexophthalmic, and twenty-five mixed, made up the total number of toxic cases. The criteria of classification corresponded approximately to those familiar as "sympathicotonic" and "vagotonic." There were no cases of pure sympathicotonia or vagotonia. The cases grouped as exophthalmic were characterized, mainly, by a marked degree of exophthalmos and of tachycardia, and by the absence of hyperhidrosis.

In the mixed group, 68 per cent. of the patients exhibited more or less exophthalmos, and 36 per cent. positive von Graefe and Stellwag signs. In 32 per cent., the pulse rate sitting was 100 or over; 52 per cent. showed well marked hyperhidrosis. In the toxic nonexophthalmic group, 15 per cent. showed a tendency toward exophthalmos, and 20 per cent. had a pulse rate, sitting, of 100 or over. Forty per cent. had typical hyperhidrosis.

Definite visible and palpable thyroid enlargement was present in approximately one half of the hyperthyroid cases, taking the three groups together; it was more frequent in the nonexophthalmic than in the others. The usual tendency toward a more marked affection of the right lobe than of the left was verified. Typical fine hyperthyroid tremor was the rule.

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20. Plummer, Henry S.: The Function of the Thyroid, Normal and Abnormal, Trans. Ass. Am. Phys. **31**:128, 1916.

21. Eppinger and Hess: Vagotonia, trans. by Kraus, Walter Max, and Jelliffe, Smith Ely, J. Nerv. & Ment. Dis. Monograph, N. Y., 1915.

Psychotic tendencies, when present, were in the direction of mild depressed states. M. S. Woodbury [22] remarks that thyrotoxic psychosis probably does not deserve a separate classification, but adds that in cases of patients not actually insane a definite mental and nervous picture does appear, differing somewhat in type according to the type of dysthyroidism present. He finds practically all patients with toxic cases "nervous" in some degree, from moderate nervous hypertension to extreme restlessness, excitability and irritability, all these symptoms being less striking in nonexophthalmic than in exophthalmic goiter. Our findings are identical in these respects. His conclusion that a depression of moderate duration is commoner in toxic nonexophthalmic than in toxic exophthalmic cases could not be verified, since that symptom was observed in only five of the ninety-three cases; these five patients, however, all had toxic nonexophthalmic goiter. Next to "nervousness," the commonest single psychoneurotic symptom was insomnia, occurring in one fifth of our series.

ETIOLOGY

An acute infection preceding the onset of hyperthyroidism, or a chronic infection concurrent with it, whether in a causal relationship or not, appeared in the histories of approximately three quarters of the series. With the exceptions of pharyngitis or tonsillitis (in six cases), influenza (in nine), and chronic bronchitis (in fourteen), no one infection was associated with more than one or two cases of hyperthyroidism. C. A. Roeder [23] reported a number of cases of toxic goiter "developing rapidly and immediately following the infection" (epidemic influenza). J. H. Carroll [18] noted that "men ill with trench fever uniformly evidenced marked vagal phenomena during the febrile and convalescent stages." Trench fever did not appear at all in our series. It is a fair assumption that many of the patients with hyperthyroid cases caused by the war, at least of the vagotonic or toxic nonexophthalmic type, have made good recoveries.

Although quite in agreement with those who ascribe etiologic importance to shell-fire and gassing, we have drawn no conclusions from the presence or absence of these causative factors on account of their uncertain history. Gas is too frequently named by the patient now as a cause for all ills, even more so than in the American Expeditionary Forces. Hyperthyroidism arising in gas warfare may be referable to gas intoxication directly, or may be part of a "gas syndrome" or "gas neurosis" (differentiated by Schwab [24]) considered psychoneurotically.

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22. Woodbury, M. S.: The Psycho-Neurotic Syndrome of Hyperthyroidism, J. Nerv. & Ment. Dis. **47**:401 (June) 1918.

23. Roeder, C. A.: Toxic Goiter Following Epidemic Influenza, Surg. Gyn. & Obst. **30**:357 (April) 1920.

24. Schwab, Sidney I.: The War Neuroses as Physiologic Conservations, Arch. of Neurol. & Psychiat. **1**:579 (May) 1919.

High explosive shelling with possibly resultant [24] "concussion syndrome," "concussion neurosis," or other psychoneurosis, which may be accompanied by hyperthyroidism, can rarely be reckoned positively present or positively absent in a given case history.

TREATMENT AND COURSE

Operations on the thyroid were performed in only eight cases, with results showing no significant difference between patients treated surgically and those treated medically. In either instance, any governmental control over treatment is uncertain at best. Many examinations, being initial during the period studied, afforded no bases for comparisons; many others, not initial, showed no net gain or loss; about one quarter of the total were marked definitely "better" (60 per cent.) or "worse" (40 per cent.), and this predominance of improvement applied to each of the three subgroups, toxic nonexophthalmic, exophthalmic and mixed. Most (78 per cent.) of the series were rated by the examiners as "feasible for vocational training," but the success of those persons who have undertaken it cannot be determined for some time.

Many factors other than strictly medical or surgical considerations will necessarily influence the further course of the disease in any one of these cases. Almost all of the patients are *a priori* psychoneurotic and prone to chronic invalidism; their youth is favorable, but their compensability may be a hindrance; even intensive handling of psychoneurotic patients, as summarized by Richards,[25] scarcely promises brilliant results.

Von Noorden [26] maintained that neither the purely vagotonic nor the purely sympathicotonic type of exophthalmic goiter was accompanied by psychopathologic symptoms; these were confined to mixed types of the disease. The more prevalent conception today is that all cases of exophthalmic goiter are more or less mixed and may be more or less psychoneurotic. In our series, the "mixed" cases are those in which neither exophthalmic (sympathicotonic) nor nonexophthalmic (vagotonic) tendencies are definitely predominant.

SUMMARY

To summarize, our material consisted of ninety-four consecutive outpatient cases, clinically examined, of war veterans suffering from thyroid dysfunction accompanied by some neuropsychiatric disorder. We conclude: (1) that the incidence of dysthyroidism, increased by

25. Richards, Esther L.: A Study of the Invalid Reaction, Arch. Neurol. & Psychiat. **2**:393 (Oct.) 1919.

26. Von Noorden, Karl: Zur Kenntnis der Vagotonischen und Sympathikotonischen Fälle von Morbus Basedowii, Kiel, 1911.

the war, is now on the decline but has not yet reached its prewar level; (2) that the bulk of this increase is made up of toxic nonexophthalmic, vagotonic cases, and cases transitional between this and the sympathicotonic type; (3) that many causes for the increase have been variously operative, none predominating; and (4) that there is a distinct tendency toward improvement in all types of toxic cases caused by the war.

A CASE OF HEREDITARY ATAXIA (FRIEDREICH) WITH ANATOMIC FINDINGS

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CASE REPORT

H. G., a man, born May 22, 1887, apparently was well during early childhood. His father died at the age of 50 from acute nephritis, his mother was living and healthy. The family consisted of one son and three daughters. One of the daughters died in early infancy, the other two were in good health; they were married and had families. Apparently his grandparents lived to an old age. While the family of one of his sisters had not exhibited any hereditary tendencies, two of the children of the other displayed significant symptoms. There was no history of syphilis or tuberculosis in the family.

The patient walked and talked at 18 months. At the age of 7 he had measles. Beginning when 6 years, he went to school for seven years and did fairly well with his studies. He then became a machinist's apprentice. When 14 years old, one afternoon he became dizzy and faint and had to be assisted to his home. He was quite nervous and later showed some ataxia, so that he did not return to work for nearly four months. During the next three years he worked, but there was considerable unsteadiness of gait. This increased so that at 18 years of age he could no longer obtain employment. He also suffered from pains and jerking movements in the extremities. The arms were weak and his hands were shaky. He was able, however, to get about for several years with the aid of a cane or crutches, but subsequently walking became practically impossible.

The patient was rather thin, 5 feet 4 inches (163 cm.) in height and weighed 119 pounds (54 kg.). His head was well shaped and inclined somewhat forward. There were no eruptions or discolorations and no glandular enlargements. The thoracic and abdominal organs and the genito-urinary system were normal. The Wassermann reaction with the blood and spinal fluid was negative. The spine showed marked dorsal curvature, convexity to the right, extending from the fifth dorsal vertebra to the first lumbar.

The pupils were normal, but turning the eyes to the side, up or down, caused irregular twitchings. When protruded, the tongue showed fine oscillatory movements. The voice was low and monotonous, and speech slurred, especially for some words. Knee and elbow reflexes were absent, the visceral reflexes intact, the plantar reflexes were exaggerated and the abdominal reflexes quite active.

The upper extremities presented no deformity; the bones were well shaped, but the muscles on the dorsal aspect of the forearm were smaller than normal. The calf muscles were poorly developed, and the anterolateral group was defective. The heel was drawn up, and the great toe of the left foot was in hyperextension. The other toes were hyperextended at the metatarsophalangeal joint and flexed at the phalangeal joints.

When sitting, there was some swaying of the head and body. Jerking movements of the head and twitching of the face muscles sometimes occurred.

The upper extremities were freely movable. Ataxia was evident. There was difficulty in picking up objects; the movement was awkward and of a wavering, jerking character. The finger to finger and finger to nose tests also revealed ataxia.

Muscular power of the lower limbs was decreased. Neither leg when extended could be raised more than 6 inches (2.34 cm.). In walking with assistance, his steps were short, the feet thrust too far forward and thrown several inches beyond the place they finally touched. The feet reached the floor with a stamp, the toes first, and then the heels. Voluntary movement at the ankle was confined to slight extension. Movement of the toes was better. Passive movement at the ankle was much more extensive.

The disease progressed slowly. Disturbance of sensibility appeared. All light contacts with a wisp of cotton were appreciated over the trunk. On the palmar surfaces of the hands and the distal parts of the fingers, some hypesthesia was present, but over the rest of the upper extremities sensation was normal. Tactile loss was marked on the feet and lower parts of the legs; it was less pronounced toward the knees and absent on the thighs. Sensation for pressure was everywhere normal and there was no analgesia nor thermoanesthesia. Muscular sense was quite defective, and he could recognize only marked differences in weight. He was unable to appreciate form correctly. Vibratory sense was impaired on the hands and wrists, and lost on the feet and lower part of the legs. There was definite loss of the sense of position in the fingers and hands. Passive movements could not be recognized unless extensive. In the lower extremities this was even more pronounced, especially in the feet.

During the latter part of his illness the patient showed considerable irritability and some impairment of judgment and reasoning. He died of lobar pneumonia, fifteen years after the onset of the nervous disease.

Postmortem Examination.—Necropsy examination was performed five hours after death. The body was well preserved, medium sized, and somewhat emaciated.

The calvarium was symmetrical, the wall thick, diploe compact. The dura was not adherent, thickened or discolored. The pia showed slight opacity over the convexity, and the cerebrospinal fluid was somewhat increased.

The brain weighed 1,150 gm. The cerebral hemispheres presented no perceptible atrophy or widening of the fissures, and the convolutional pattern appeared good. The basal vessels were normal. The lateral ventricles were not dilated, and no ependymitis was in evidence. The membranes of the spinal cord were slightly thickened and the cord diminished in size. At different levels the posterior columns were seen to be affected.

The pericardium was normal and contained clear fluid. The heart weighed 220 gm. The right auricle, tricuspid orifice and valves appeared normal. The right ventricular wall was slightly thinned. The cavity was normal. The pulmonary valves and artery were normal. The left auricle was somewhat enlarged. The mitral leaflets were slightly thickened. The left ventricular wall was somewhat thickened; the aortic valve appeared normal. The right lung weighed 710 gm., the left 690 gm. Edema and areas of consolidation were present in both lungs.

The liver weighed 1,400 gm. The surface was smooth, the capsule was somewhat thickened and some passive congestion was present. The gallbladder was normal. The spleen was enlarged, the capsule thickened; the pulp was soft and red in color. The pancreas, stomach and intestines appeared normal.

The right kidney weighed 120 gm.; the left, 130 gm. The capsule stripped readily, the surface was smooth, the cortex and medulla were normal. The bladder showed nothing remarkable.

Microscopic Study.—Numerous pieces from the brain, spinal cord and peripheral nerves were examined by various staining methods: the Nissl, toluidin blue, thionin, Marchi, Weigert, Mallory, Bielschowsky, Mann-Alzheimer, hematoxylin-eosin, sudan III, cresyl-violet, polychrome methylene blue and others.

Sections from the midbrain through the crura cerebri, corpora quadrigemina, geniculate bodies and rubrum, stained by the Weigert-Pal and Marchi methods, revealed no pathologic changes.

Sections from the pons showed no alterations other than slight involvement of a few medullated longitudinal fibers.

In the medulla oblongata, degeneration was present in some fibers of the pyramids. The fibers of the olivary eminence were normal. There was no abnormal change in the fillet or dorsal longitudinal bundle. Atrophy of a considerable number of the fibers of the corpora restiforme was found. The inferior olivary nucleus and the motor nuclei were normal.

In the vicinity of the cuneate and accessory cuneate nuclei, products of degeneration were discernible. An area of degeneration was found in the funiculus cuneatus, involving the cuneate nucleus. Fibers in the nucleus gracilis were markedly atrophied. Near the upper part of the pyramidal decussation, many fibers of the funiculus gracilis and cuneatus were extensively involved. A few degenerated fibers were seen scattered through the lateral and anterior white matter. At a somewhat lower level, pronounced atrophy of the fibers of the funiculus gracilis was found and also a considerable number of degenerated fibers in the funiculus cuneatus. Other parts of the white matter also contained a few degenerated fibers.

Cellular changes in the pons and medulla were not pronounced, but the glia in the region of the pyramidal tracts appeared to be slightly increased. A few nerve cells exhibiting chromatolysis and nuclear alterations were encountered in the nucleus ruber. In the restis disintegrated cells were discernible, and a moderate degree of gliosis.

In sections from the first and second cervical segments of the cord, a narrow strip of apparently healthy fibers was seen between the posterior gray commissure and horns and the area of degeneration in the posterior columns. The column of Goll appeared to be more extensively affected than the column of Burdach. A deficiency of fibers occurred in the region of the crossed pyramidal and spinocerebellar tracts. Degeneration in the direct pyramidal tract was less pronounced. Products of degeneration and granular cells usually occupied the diseased areas; other zones were free.

In the fifth and sixth cervical segments the narrow band of unaltered fibers mentioned in the foregoing was present anterior to the diseased zone and in contiguity to the area of entry of the posterior roots. Degeneration in the columns of Goll and Burdach was well marked, but somewhat less extensive in the latter. The crossed pyramidal tracts were considerably affected with slight involvement of the direct. A moderate degree of fiber degeneration occurred in the posterior part of Gowers' tract. Atrophy of the fibers in the dorsal spinocerebellar tract was well defined.

At the fourth dorsal segment the fibers of the posterior columns were severely degenerated, but a zone of apparently healthy fibers divided the area of degeneration from the entering fibers of the posterior roots and dorsal horns.

The fiber atrophy in the lateral pyramidal tracts was similar to that described in the foregoing. Changes in the dorsal spinocerebellar tract were more pronounced than in the ventral. Medullary rests and lipoid granules dispersed through the degenerated area of the posterior columns were seen in sections stained according to Marchi's method.

In the mid-dorsal region the posterior columns were intensely diseased, especially in the interior of the degenerated area, but they were less diseased in the surrounding zone. A zone of unaffected fibers was interposed between the degenerated area and the dorsal horns. Atrophy in the right pyramidal tract appeared more extensive than in the fourth dorsal segment. The dorsal spinocerebellar and lateral pyramidal tracts showed many disintegrated fibers. At the level of the tenth dorsal roots, marked degeneration obtained in the dorsal columns. The direct spinocerebellar tract was somewhat altered, and the lateral pyramidal tracts considerably affected. In the lumbar region the posterior columns were extensively involved, with considerable change in the lateral pyramidal tracts and some disintegrated fibers in the dorsal spinocerebellar tracts.

With neuroglia stains, a thick netlike structure of varying density was found replacing the atrophied nerve fibers. Within the distended meshes of the network swollen medullary sheaths and axis cylinders were discernible. The proliferated glial fibers usually were homogeneous. Glia cells with pale enlarged nuclei and granular nucleoli and large stellate cells with swollen irregular nuclei were more or less numerous.

In the anterior and posterior horns a few cells showed chromatolysis. A few cells were shrunken, elongated and intensely stained; some were swollen, irregular and showed disintegration of the tigroid substance. Increased pigment and eccentricity of nuclei were seen and occasionally regressive changes. There was slight glial increase. Cells of Clarke's column were badly disintegrated at all levels, with marked proliferation of neuroglia and obliterated capillaries. The anterior roots were normal. The posterior roots exhibited atrophic and disintegrative changes, more pronounced in the lower segments.

In the frontal region of the cerebrum changes were found in the pyramidal layer. Nuclei of cells were enlarged and contained numerous faintly stained granules. In the vicinity of the nuclei the cytoplasm presented a netlike structure, but in the periphery and apical processes was filled with numerous lightly stained particles. Some pyramidal cells were swollen, pale and rounded, the bodies and processes being entirely filled with pale, ill-defined granules, among which irregular, darkly stained masses were frequently distributed. The nuclei were distended and the nuclear membrane delicate, being at times hardly distinguishable from the cytoplasm. The caryoplasm contained fine granules similar to those in the cytoplasm. The axis cylinder processes were unaffected and the neurofibrils well preserved. No neuroglial proliferation was present but progressive gliogenic changes were seen in the vicinity of many ganglion cells. Lipoid granules were not infrequently encountered within the adventitial spaces and cells of the cortical vessels.

In the precentral gyrus the cytologic alterations were slight. Some of the large motor cells were somewhat affected in their staining and morphologic characteristics. Considerable increase in pigment and tigrolysis occurred. The neurofibrils showed no marked alterations. Changes were discernible in many of the associative and commissural neurons. Some cells were swollen and the chromatic substance was lightly stained. In others chromatolytic changes were in evidence and fine dust-like particles were scattered irregularly through the

cell. The chromophilic bodies were sometimes aggregated in masses or appeared elongated. Almost complete disappearance of the Nissl substance occurred in some cells, which were reduced to shadows. Shrinkage of the achromatic substance was observed in cells showing degenerative changes, and the tigroid bodies within the cell body and the dendritic processes were more deeply stained. Such cells occasionally appeared elongated as a result of the excessive shrinkage. The nuclei were frequently deformed and dislocated. Increase of glia was proportionate to the changes in the ganglion cells. Satellitosis was present in the vicinity of nerve cells, and glia cells were occasionally seen within the cytoplasm of the altered ganglion cells.

Changes in the postcentral and paracentral gyri were similar to those in the precentral convolution. The basal ganglions revealed no significant alterations of cells or fiber content.

In the corpus dentatum of the cerebellum some of the large triangular and stellate cells were shrunken and atrophic. A few Purkinje cells showed chromatolysis, and neurofibrillar disintegration was perceptible. In the granular layer some cells appeared shrunken, deeply stained and their processes unusually tortuous. Eccentricity of nuclei with pigmentary increase was frequent. Fiber decrease in some areas was seen by using the Bielschowsky and Weigert-Pal methods. In areas of cellular alteration, changes in the neuroglia were evident: increase of glia cells and hyperplasia of neuroglia fibers. The adventitial spaces of capillaries in these zones contained particles of detritus, but no endarteritic changes were perceived.

The peripheral nerves investigated were the ulnar, radial, musculospiral, anterior and posterior tibial, anterior interosseous and sciatic. Atrophic fibers were found in some sections. Most of the fields contained a normal fiber content. In the larger nerves it appeared that a greater degree of atrophy obtained in the peripheral portions.

SENSORY CHANGES IN FRIEDREICH'S ATAXIA

Cases of Friedreich's ataxia have been reported with loss to different forms of sensibility. A number of authors have recorded sensibility as intact, although degeneration of the posterior columns was found in all cases anatomically studied. The preservation of sensation with pronounced degeneration of the posterior columns, seemingly a paradox, has been variously explained.

Friedreich [1] averred that the dorsal columns could not be the principal sensory tracts. Müller [2] thought that sensory impulses were conducted by naked axis cylinders within the degenerated tracts. Déjerine [3]

1. Friedreich: Ueber Ataxia mit besonderer Berücksichtigung der hereditären Formen, Virchows Arch. f. path. Anat. **26-27**:391, 1863; Ibid. **68**:145, 1876; Ibid. **70**:140, 1877.

2. Müller: Zur Pathologie der Friedreichschen Krankheit, Deutsch. Ztschr. f. Nervenheilk. **32**:137, 1907.

3. Déjerine: Sur une forme particulière de maladie de Friedreich avec atrophie musculaire et troubles de la sensibilité, Méd mod. **1**:477, 1890. Les lesions des racines, des ganglions rachidiens et des nerfs dans un cas de maladie de Friedreich, Rev. neurol. **15**:41, 1907.

believed that sensation was not affected by degeneration in the columns of Goll, and that alterations in the posterior columns may exist without sensory disturbances.

Mott [4] claims that the neurons consisting of small cells and fine fibers, which subserve cutaneous sensory and viscero-vascular functions, are not affected in Friedreich's disease as in tabes, and in consequence there are no visceral disturbances or loss of cutaneous sensibility.

While extensive involvement of the nervous system together with disease of other organs of the body is frequently found in Friedreich's ataxia, the changes in the posterior columns are the most definite and invariable feature. The condition is considered essentially as a systemic degeneration of the dorsal columns. In the upper part of the cord the degeneration is not so pronounced, but in the lumbar and sacral regions the degeneration involves almost completely the dorsal column. In the cervical and dorsal regions, the fasciculus marginalis of Lissauer and the dorsal cornu commissural tract, which occupies a triangular interval at the apex of the transsected dorsal column, are practically free from the degenerative process. With these exceptions the dorsal column is intensely degenerated.

It is obvious that the lesion is principally of the long and medium fibers of the posterior column. Degeneration in the pyramidal and spinocerebellar tracts varies in different cases, but seldom equals that of the dorsal column. Involvement of the spinocerebellar tracts is greater in the upper dorsal and cervical regions, whereas the pyramidal tracts are less degenerated in the higher segments. Pyramidal tract degeneration may extend into the midbrain. Occasionally the direct pyramidal tract is slightly affected.

Degeneration of the ganglion cells of Clarke's column occurs; otherwise cellular changes are slight. The muscle fibers and peripheral nerves are sometimes affected. In view of the slight or moderate alteration in the spinal ganglion cells and posterior roots, the degeneration in the dorsal column could hardly be considered as consequent on primary degeneration of the posterior roots and ganglion cells.

The pathologic anatomy of hereditary ataxia, therefore, exhibits extensive system lesions, involving the long spinal tracts to some degree but chiefly those of the posterior column. Obviously sensory impulses transmitted by the affected tracts should be disturbed.

There is evidence that the column of the cord, which is profoundly degenerated in Friedreich's disease, conveys impulses for the recogniza-

4. Mott: Arch. Neurol. from the Path. Lab. of the London County Asylums **3**:180, 1907.

tion of form,[5] size and weight,[6] passive movement and position, compass points applied to the skin [7] and vibration.[8]

It is generally conceded that impulses of deep sensibility utilize two routes to the brain. One is by the long fibers of the dorsal roots and posterior tracts to the optic thalamus and cerebral cortex, ascending uncrossed; the other by the median fibers of the posterior roots, column of Clarke and the dorsal spinocerebellar tract, in the dorsal periphery of the lateral column of the same side to the medulla, thence by the restiform body to the cerebellum, terminating in the vermis. The cerebellar fibers subserve subconscious tactile and coordinatory perceptions, while the cerebral make possible conscious perception of position and motion.

Painful and thermal sensations are conducted by short fibers of the dorsal root system to cells in the posterior horns, thence by fibers of the spinothalamic tract, decussating by way of the anterior commissure and terminating in the optic thalamus.

In my case there was definite loss of the sense of position and of passive movement (conveyed chiefly by the posterior column), with disturbance of vibratory sense and considerable loss in the recognization of weight, form and size. Tactile sensibility was slightly affected over the distal parts of the extremities, but thermal and pain senses were intact.

A number of cases are recorded which exhibit similar sensory disturbances. The loss in sensibility may be difficult to demonstrate in the initial stage. In some cases motor disability appears to be less pronounced, compared with the degree of sensory loss, than we might expect, and *vice versa*. There are advanced cases presenting only moderate ataxia, while others are markedly ataxic, even in the early stages.

A study of the pathology apparently warrants the conclusion that when the forms of sensibility transmitted by the dorsal column are impaired with only moderate ataxia, the degeneration affects principally the posterior tracts; but when sensory loss is slight in proportion to ataxia, the spinocerebellar tracts are affected more intensely than the dorsal column tracts. Many variations therefore occur, depending on the regional preponderance of degeneration. Sensory alteration is invariably first found in the lower extremities, is distal in distribution, usually

5. Head and Holmes: Sensory Disturbances from Cerebral Lesions, Brain **34**:102, 1911-1912.

6. Head and Thompson: The Grouping of Afferent Impulses Within the Spinal Cord, Brain **29**:537, 1906.

7. Head, Rivers and Sherren: The Afferent Nervous System from a New Aspect, Brain **28**:99, 1905.

8. Bing: Vibrationsgefühl, Neurol. Centralbl. **29**:173, 1900.

symmetrical, in the later stages of the disease quite marked, contributing largely to the disability of the patient. The sensory disturbances in my case correspond to the neuropathologic findings, and both conform to the general conception of sensory conduction in the cord. I believe that the characteristic sensory loss in Friedreich's ataxia is an essential rather than an exceptional feature in the symptomatology of the disease.

TWO RECENT IMPROVEMENTS IN THE STAINING OF SPIROCHETES IN NERVOUS TISSUE

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Since 1916 several important contributions to the study of neurosyphilis have appeared in the German literature. These have been based in part on studies of spirochetes made possible by Jahnel's [1] modification of the method of silver impregnation. Little, if any, reference has been made to these contributions in our American literature. This is probably due to the failure of the periodicals to reach the United States, and to failure or delay in indexing. It is hoped that by here presenting Jahnel's method it will be more accessible and better appreciated. Comment, such as is justified by a short acquaintance with, and use of, the method, will be made.

Similarly the recently published cover-glass method of Warthin and Starry [2] will be referred to.

JAHNEL'S METHOD

The failure of foreign workers to obtain satisfactorily constant results with existing silver impregnation methods in nervous tissue led Jahnel to evolve his method. Our experience with the method in the laboratory of the Psychiatric Institute has been highly satisfactory. The technic, while requiring a long time, offers few pitfalls if the directions are followed. The results are not only very useful, but actually beautiful. With a subdued yellow to yellowish-brown background the spirochetes stand out in marked contrast. The staining of nerve fibers is so suppressed as to eliminate any confusion with spirochetes. Granulation, fraying and precipitate are negligible. While in our experience no old chemicals have been used, Jahnel warns against these. He offers an alternate developer,[3] but owing to the success obtained by using the simpler one, we have not tried the other. No modification of the method has been found necessary. Fixation should be complete; our most beautiful preparations were made with tissue that had been in formaldehyd ten years. In step 1, it is desirable to wash under sterile conditions.

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1. Jahnel, F.: München. med. Wchnschr. **67**:932 (Aug. 6) 1920.
2. Warthin, A. S., and Starry, A. C.: Second Improved Method for the Demonstration of Spirochaeta Pallida in the Tissues, J. A. M. A. **76**:234 (Jan. 22) 1921.
3. Jahnel, F.: München. med. Wchnschr. **67**:1263 (Oct. 29) 1920.

JAHNEL'S METHOD FOR STAINING SPIROCHETES IN THE BRAIN

1. Wash out in water, in from one to three days, thin pieces from 2 to 4 mm. fixed in formaldehyd or alcohol.

2. Place in pure pyridin one to three days.

3. Wash in many changes of water until the pyridin odor almost disappears during a period of from two to three days; this is important. Then allow the pieces to remain a "few days" (einige tage) in a 5 to 10 per cent. formaldehyd solution, U. S. P.

4. Place in water again. (The time in water this time is not stated; probably the washing should be thorough.)

5. Treat with uranium nitrate (Merck), 1 per cent. solution in distilled water, one-half to one hour in the incubator at 37 C. The use of glass wool (lead free) under the tissue helps the penetration but is not absolutely necessary. The purpose of the uranium nitrate is to prevent the coincident staining of other elements of the nervous tissue.

6. Wash out in distilled water for one day.

7. Allow to remain in 96 per cent. alcohol three to eight days.

8. Wash out in distilled water until the block sinks.

9. Place the tissue in a dark (amber) flask and treat it with a freshly prepared silver nitrate solution, 1.5 per cent., five to eight days in the oven at 37 C.

10. Decant the silver nitrate solution; wash the tissue slightly in water; then transfer it to a solution made up as follows:

	c.c.
4 per cent. aqueous solution of pyrogallol	95
Formaldehyd solution, U. S. P.	5

Allow the tissue to remain in the solution one to two days.

A later note states that, especially with thicker pieces and fresher material, short washings in water after the silver nitrate treatment and the use of the pyrogallol-acetone-pyridin developer is to be recommended. This developer is made as follows:

	c.c.
4 per cent. pyrogallol	90
Acetone	10

From this decant 15 c.c. and replace by pyridin.

11. Wash out in distilled water; embed in paraffin.

COVER-GLASS METHOD

The cover-glass method of Warthin and Starry, as originally published was not specifically adapted to nervous tissue. Treating the sections with uranium nitrate was given as one alternate in combating the failure of the tissue to stain properly. However, with nervous tissue, this treatment with uranium nitrate seems uniformly necessary. The cover-slip method offers two advantages: It is rapid, and it permits adjacent sections to be examined by other stains. On the other hand, it has the disadvantages of making the tissue somewhat granular, of staining the nerve fibers, although not seriously, and probably breaking up some of the spirochetes so that if few are in the section, the

chance of finding them is reduced. Thus negative findings are inconclusive. After the paraffin mounts have been made (this may be done when cutting sections for the other stains), the method requires about one and one-half hours. From thirty minutes to one hour of this time is spent in impregnation, so that the work required actually takes less time. The main pitfall lies in developing. In preparing the developer it is necessary to work rapidly, to stir in the hydroquinone as quickly as possible, and to immerse the cover-slips edge up, since it is but a minute or two until the silver is reduced and the developer changed to an opaque mass. From this mass the cover-slips must be removed in order to determine whether the stain is sufficiently reduced. A yellowish-brown color is most desirable. In view of the fragility of the spirochete, it is further desirable to avoid too rapid hydration or dehydration. On this account we have suggested the graded alcohols in place of absolute alcohol.

COVER-GLASS METHOD

1. Fix completely in 10 per cent. formaldehyd solution, U. S. P., pieces from 5 to 7 mm. thick.

2. Embed in paraffin without washing in water.

3. Cut from 5 to 12 micron sections and mount on specially prepared cover-slips with minimum albumin fixative (three-fourths inch No. 1 coverslips, cleaned thoroughly and dried in the incubator for twelve hours).

4. Remove the paraffin. (Warm over a flame, wash with xylol, descending alcohols and water.)

5. Immerse the section in 1 per cent. uranium nitrate (distilled water) five to eight minutes.

6. Wash in distilled water one minute.

7. Rinse the section and a clean cover-glass in fresh silver nitrate solution, 2 per cent. Apply the rinsed cover-glass to the section so that they will adhere by capillary attraction, and put edge up and against the side in a dark bottle containing half an inch (1.27 cm.) of fresh 2 per cent. silver nitrate solution; cork tightly. Incubate from thirty to sixty minutes in the dark.

8. Separate the cover-slips and put the section into the following reducer. Remove when yellowish to reddish brown.

Mix 2 per cent. silver nitrate 3 c.c., warm 10 per cent. aqueous gelatin 5 c.c., warm glycerin 5 c.c.

With stirring add warm 1.5 per cent. agar suspension, 5 c.c. (The agar suspension is made by mixing 1.5 gm. agar with 100 c.c. cold distilled water, heating slowly, and stirring constantly until it boils. Continue to boil until suspension is complete. Keep on top the paraffin oven.)

With stirring add a 5 per cent. aqueous hydroquinone solution 1 to 2 c.c. just before immersing sections.

9. Rinse the section in a 5 per cent. solution of sodium thiosulphate.

10. Rinse the section in distilled water.

11. Treat with ascending alcohols, xylol; mount it in balsam.

A STUDY OF SINISTRALITY AND MUSCLE COORDINATION IN MUSICIANS, IRON WORKERS AND OTHERS

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INTRODUCTION

In a previous communication,[1] based on a survey of forty-two sinistrals,[2] the idea was advanced that the disorders of speech so often noted in left-handed people are due to congenital inferiority of the central nervous system. If this notion is valid, it would be logical to look for evidence of faulty team-work in various functionally related muscles, for there seems to be no especial reason why, in sinistrals, incoordination should be limited to the muscles of the speech group. Accordingly, a tentative study of sixteen sinistral private patients was made, with the following results: Six admitted an unaccountable and capricious tendency to "bump into things." One is unable to "reverse" in waltzing. Of five who had studied instrumental music, two had made fair progress up to a certain point but considered that they had accomplished little for the outlay of time and money. Both are hampered by a defective sense of tempo and find "sight-reading" an embarrassment. Two others abandoned the study of music because they were "unable to make any headway." The remaining one is a talented player but states that she still finds it difficult to "keep in time." Of these five musicians, two are left-handed and left-eyed, one is left-handed and right-eyed and two are right-handed and left-eyed. Five patients are free from motor symptoms.

These patients seem to show: (1) that sinistrals are especially prone to various forms of muscle incoordination, and (2) that in some of these persons both the sense of equilibrium and the sense of rhythm are defective. Hoping further to elucidate the subject, a study of sinistrality in skilled manual workers was carried out.

MATERIAL

Three series of 100 men each, classified as (1) professional musicians, (2) machinists, and (3) male inmates of a public relief home, were examined.

1. Quinan, C.: Sinistrality in Relation to High Blood-Pressure and Defects of Speech, Arch. Int. Med. **27**:256, 1921.

2. Throughout this paper the word sinistrality is used to designate partial as opposed to established left-sidedness. The most common symptoms of this condition are dominance of the left eye in binocular vision and a limited use of the left hand as the leading hand.

Professional Musicians.—First, players in five orchestras, all members of the musicians union, were examined. The men were tested without selection except that players on stringed instruments were especially sought as representative of the highest product of coordinated muscle activity. They constitute 64 per cent. of all musicians examined. Pianists were put in second place and a few players on wood-wind and brass instruments make up the total. The piano group includes a few unattached professional virtuosi.

It was thought that successful instrumentalists would prove to be almost exclusively dextral, because it seemed likely that left-handed and sinistral persons would instinctively seek a livelihood in some less exacting vocation. Unexpectedly it was found that one in every three musicians could be classified as left-handed or sinistral. Therefore it became necessary to select a control group, and machinists were chosen because in addition to at least average intelligence they offer the desirable combination of trained eyes and powerful muscles.

TABLE 1.—ANALYSIS OF ONE HUNDRED PROFESSIONAL MUSICIANS WITH REFERENCE TO SINISTRALITY

	Number Examined	Right-Handed		Left-Handed		Total Sinistrality Percentage
		Dominant Eye		Dominant Eye		
		Right	Left	Right	Left	
Violin and viola	40	27	11	1	1	32.5
Cello and string-bass	24	17	5	0	2	29.1
Piano and organ	18	13	3	1	1	27.7
Flute and oboe	10	8	2	0	0	20.0
Trombone	8	3	3	0	2	62.5
Totals	100	68	24	2	6	32.0

Machinists.—Workers in three large machine shops were chosen. The men were examined without selection. A few apprentices and a small number of iron-workers who had not had the training of a machinist are included in the series.

Relief-Home Inmates.—Finally, a number of old men in a public relief-home were examined. Naturally a good many of these old men were physically infirm and probably of rather low average intelligence, but any man was considered acceptable if he had free use of his extremities together with relatively normal binocular vision.

METHOD OF TESTING

Information was sought from each person as to (1) handedness and (2) eyedness.[3] In musicians, speech defects also were noted and performance tests were made to determine the relative skill of the right

3. Reference here is made to the fact that in normal binocular vision nearly every person has a dominant or "fixing-eye."

and left hands. Here it will suffice to outline the performance test, as the method of determining handedness and eyedness has been described in detail elsewhere.[1]

Performance Test.—For this purpose I used the ordinary "primary-pegging-board." This consists of a thin piece of board, 10 inches (25.4 cm.) square, one side of which presents 100 shallow holes

TABLE 2.—PERFORMANCE TESTS OF THE RIGHT AND LEFT HANDS IN THIRTY-TWO LEFT-HANDED AND SINISTRAL MUSICIANS

No.	Dominant		Pegging Board Scores		History of Stammering	Faulty Rhythm	Notes
	Hand	Eye	Right	Left			
1	L	L	37	46	Yes	Yes	Strongly left-handed
2	L	L	44	49	Yes	....	Strongly left-handed
3	L	L	50	55	Yes	Yes	Strongly left-handed
4	L	L	32	36	....	....	Strongly left-handed
5	L	L	40	34	....	....	Trombonist; writes with left hand
6	L	L	40	48	Yes	....	Strongly left-handed
7	L	R	38	56	Yes	....	Strongly left-handed
8	L	R	31	45	....	....	Strongly left-handed
Aver.	..	..	39	46			
9	R	L	46	34			
10	R	L	49	39	Yes	....	Deals cards with left hand
11	R	L	63	44			
12	R	L	36	31	....	Yes	Deals cards with left hand
13	R	L	46	46	Yes	....	Deals cards with left hand
14	R	L	51	49	Yes	....	Gun at left shoulder, aims with left eye
15	R	L	49	32			
16	R	L	37	37	....	....	His only child is left-handed
17	R	L	48	42	....	....	Deals cards with left hand
18	R	L	30	31	Yes	....	Deals cards with left hand
19	R	L	48	40	....	Yes	
20	R	L	51	43	....	....	Gun at right shoulder, aims with left eye
21	R	L	25	29	....	....	Years ago slight right hemiplegia
22	R	L	45	42			
23	R	L	46	36			
24	R	L	43	38	....	....	Holds spoon, wields axe left-handed
25	R	L	49	36			
26	R	L	38	36	Yes	....	Gun at right shoulder aims with left eye
27	R	L	43	45	....	....	Deals cards with left hand
28	R	L	54	41			
29	R	L	40	30	....	....	Gun at right shoulder, aims with left eye
30	R	L	38	34			
31	R	L	44	38	....	....	Gun at right shoulder, aims with left eye
32	R	L	46	42	....	....	Deals cards with left hand
Aver.	..	..	44.3	38.1			

arranged in ten parallel rows. Wooden pegs are provided with the board. In carrying out the test, in a time allowance of one minute, as many pegs as possible are set up by the right hand always moving to the right. The left hand is tested in like manner but moves toward the left. A peg duly placed, that falls, counts. Although the score of each hand improves with practice, the ratio usually remains fairly constant so that a single trial of skill imposes a uniform handicap.

TABLE 3.—RELATION BETWEEN MANUAL AND OCULAR DOMINANCE IN IRON-WORKERS, RELIEF-HOME INMATES AND PROFESSIONAL MUSICIANS

	Nominally Right-Handed		Nominally Left-Handed		Total Sinistrality A+b+c
	Dominant Eye		Dominant Eye		
	Right	Left (a)	Right (b)	Left (c)	
100 machinists and iron-workers	92	4	0	4	8
100 relief-home inmates (male)	78	17	1	4	22
100 professional musicians	68	24	2	6	32

DISCUSSION OF RESULTS

Briefly stated, the results show: 1. It is possible by a performance test to differentiate established left-handedness from the lesser grades of sinistrality. 2. Sinistrality is extremely prevalent in musicians as compared with other skilled manual workers. 3. Sinistral musicians are prone to faulty rhythm and other psychologic disturbances.

DIFFERENTIAL VALUE OF THE PERFORMANCE TEST

(*a*) *Individual Peculiarities.*—Musicians who try this test, even though they make nearly equal scores, exhibit widely different grades of native dexterity; one will make an average score with much fumbling and waste of energy, another will score a like number with the utmost economy of effort. Awkwardness is more characteristic of sinistrals than of dextrals. Indeed two sinistral musicians (Nos. 12 and 18, Table 2) recall Sir Thomas Browne's [4] description of people who are "ambilevous or left-handed on both sides; such as with agility and vigour have not the use of either: who are not gymnastically composed: nor actively use those parts. Now in these there is no right hand." Right-handed people invariably score more with the right than with the left hand. Just the opposite is true of those who are predominantly left-handed. But it is surprising that many musicians who use the left hand rather freely—say in dealing cards, or in other usually right-handed activities—run up average dextral scores. As far as my experience enables me to judge, such men as a rule are left-eyed. Orchestra leaders and musicians of acknowledged virtuosity almost always make scores that are close to, or often a little below, the average. An extremely high score seems to indicate nervousness rather than technical skill. Age, on the other hand, affects the score unfavorably. The idea that the magnitude of a player's score may bear some relation to the technical difficulties of his instrument is not borne out by the tests. On this point the performance scores leave no doubt.

4. Browne, Sir Thomas: The Works of Sir Thomas Browne, edited by Charles Sayle, Edinburgh, John Grant **2**:122, 1912.

(*b*) *Differential Scores.*—Forty violin and viola players made an average score of 47.8 for the right hand and 41.1 for the left hand. The corresponding figures for twenty-four cello and string-bass players are 47 and 37.4 and for eighteen piano and organ players 47.6 and 40.4. Obviously these group differences are negligible. Of somewhat greater interest is a comparison of the dextral and sinistral scores.

In the violin group, thirty-eight of the players are right-handed men. Divided with reference to the "master-eye," twenty-seven of the men are right-eyed and eleven are left-eyed. A glance at the performance scores of these two groups shows a slight advantage on the side of the dextrals, as the figures for the right-eyed group are 49 for the right hand and 41.7 for the left hand, while for the left-eyed group the figures are 46.7 and 38, respectively. The difference certainly is not striking, but it is suggestive when considered in connection with the fact (Table 2) that twenty-four sinistral players, all instruments included, have an average score of 44.3 for the right hand and 38.1 for the left hand.

The eight left-handed musicians made an average score of 46 for the left hand and 39 for the right hand. This, it will be noted, is almost exactly the reverse of the right-handed score.

Practically speaking, then, the results show that it is possible to diagnose established left-handedness by means of a simple test. But it is also possible to go farther than this and to utilize this test in the differentiation of left-handedness and sinistrality. This of course is a matter of classification. In the work here reported, it was found convenient to divide the material into the following three groups: (1) The dextral group. This includes those who are both right-handed and right-eyed, with no trace of left-handed initiative. (2) The left-handed group. Predominant left-handedness here is characteristic and the typical performance score is the reverse of the dextral. The "master-eye" may be right or left. (3) The sinistral group. Actually, right-handedness predominates in this group, but some form of left-handed initiative is almost always present and there are many instances of so-called "ambidexterity." Some of these men consider themselves left-handed. The members of this group are dextrals by the performance test, but usually the left eye is the "master-eye."

At this point a case may be mentioned showing that a comparative test of manual dexterity may have useful clinical applications. The patient was a player of a stringed instrument (No. 21, Table 2) actively engaged in professional work. He proved to be right-handed and left-eyed, in other words, a "sinistral"; but, strangely enough, he made not only a low score with either hand but his score was also typically a left-handed one. These anomalous findings at once became clear when

he made known that he had had some years previously a slight "stroke" affecting the right side.

PREVALENCE OF SINISTRALITY IN MUSICIANS

Four per cent. of the machinists proved to be left-handed (Table 3) and an additional 4 per cent. had sinistral peculiarities. In striking contrast to these figures, 8 per cent. of left-handedness was found in the series of musicians while the lesser forms of sinistrality mounted to the remarkable total of 24 per cent. Such an outstanding psychologic disparity between two groups of men in whose vocations a high order of muscle coordination is essential at once raises the question: Why is left-handedness twice and sinistrality six times more prevalent in one than in the other? I shall return to this point after an attempt to determine (*a*) whether left-handed musicians enjoy a physical advantage in comparison with right-handed players, and (*b*) whether instrumental technic modifies ocular dominance.

(*a*) Is left-handedness a physical advantage? In the series of professional musicians, sixty-eight are dextrals and thirty-two either left-handed or sinistral. Every grade of technical proficiency is represented in the 100 musicians, and seventeen enjoy high repute as soloists. It is significant, however, that only one of the soloists, an organist (No. 24, Table 2), is a sinistral. Twice I have heard left-handed musicians remark that they had "had to work hard" for their music, and from conversations on the subject with a number of others I get the impression that, in the acquisition of technic, they have had to overcome greater physical odds than dextrals. The fact that in the performance test the average sinistral score is lower than the average dextral score supports this idea. Such as it is, therefore, the evidence goes to show that muscle coordination is more highly organized in right-handed players and that left-handedness constitutes no physical advantage.

(*b*) Does instrumental technic modify ocularity? Left-eyed dominance is a common sinistral symptom in musicians. One might be led to believe from the figures in Table 1 that this peculiarity occurs with varying frequency in players of different instruments, but I believe these inequalities will disappear with more statistical material. It is true, however, that some musical instruments more than others require extensive training of the left hand. For example, one might infer that the difficulties of intonation in violin playing would have a tendency to concentrate attention on the left hand and on the left eye because this eye commands a more direct view of the strings. In fact, however, vision plays a very subordinate rôle in the technic of stringed instruments. It is safe to say that even in complicated phrases the finger movements are guided almost entirely by muscle and tactile memories.

But if it were true that violin technic fosters left-eyedness, the contrary should be characteristic of an instrument like the trombone, in the technic of which the left hand is almost inactive, tone modulation being produced through free movements of the right arm and hand. Yet five of the eight trombone players examined were definitely left-handed or sinistral. This seems to show that ocular dominance is uninfluenced by technical training. But left-handedness is a physical handicap. Why, then, are there so many sinistral musicians?

Of the three classes of men under consideration, from the standpoint of physical development the machinists easily take first place. The musicians come next because age disqualifies the relief-home group. But as most of these old men in their earlier years were active in various trades or as sea-faring men, it is probable that a leveling of the age element would force the musicians into third place. Such a readjustment, moreover, would be in harmony with the comparative sinistrality data of these three classes; namely, machinists, 8; relief-home inmates, 22, and musicians, 32. According to these figures sinistrality is a rare symptom in muscular hand-workers and common in artists. Thus an intimate relation appears to exist between left laterality and a type of mental organization prevalent in musicians. I would suggest that this is not a fortuitous association, but that both left-handedness and sinistrality usually are indicative of the psychopathic constitution. To be adduced in support of this view are these facts: 1. Left-handedness is hereditary. 2. It is probably due to defective organization in the central nervous system because of the concomitant occurrence of speech defects. 3. As several writers emphasize, it is common in children of low grade mental type. It follows that a high proportion of left-handedness in any class of the population is at least suggestive of mental instability and, I believe, of a peculiar susceptibility to the influence of music. Hence sinistrals feel drawn to the musician's vocation in spite of the fact that it is a vocation for which in many instances they are not well adapted.

PSYCHOLOGY OF LEFT-HANDEDNESS

It is coming to be recognized that disorders of speech are as common in the left-handed as they are rare in dextrals. But the fact seems to have been overlooked that such disorders also are common in those ostensibly right-handed people who are left-eyed. According to the figures in Table 2, the values in left-handed and sinistral musicians are about 60 and 20 per cent., respectively. A natural inference would appear to be that these clinical forms are essentially alike and differ in degree only. The left-handed man is prone to stammer and so is the right-handed man if he happens to be functionally cross-wired and

uses his left eye for fixation purposes. Stier [5] is frankly noncommital as to the significance of left-eyedness. Citing Enslin, who experimented with right-handed and left-handed soldiers and found that while in the former the right eye was always dominant, in the latter the left eye was dominant in twenty out of fifty-eight, Stier observes: "Personally I myself make an exception, since, notwithstanding that I have excellent vision in both eyes, in the test referred to I have found in myself a preponderance of the left eye in spite of marked right-handedness. Further experiments with other persons have shown me that it is by no means easy to understand what this experiment (determination of the master-eye) indicates. A repetition of these tests in well-educated people rather than in soldiers is therefore to be recommended before we seek to explain this remarkable phenomenon." [6]

But other central nervous peculiarities occasionally are noted in musicians with left-sided dominance, and one of these is inability to solve problems in rhythm. Some of these men cannot play in perfect tempo. Unequivocal evidence on this point was obtained from four musicians (Table 2). Probably this shortcoming is more prevalent than these figures indicate as, for obvious reasons, musicians are apt to be reticent on this subject. None of the dextral musicians gave evidence of faulty rhythm. Still another manifestation of left laterality concerns the mental outlook. I have been strongly impressed by the fact that the left-handed and sinistral musicians are much more nervous and "temperamental" than the dextrals. Talkativeness, mutism, motiveless laughter, borderline depression states and in one instance a history suggestive of dual personality have been noted. Curious defense reactions arise through the inability of these persons to adjust themselves to a right-handed environment. They constitute a class of "square pegs" whose mental difficulties would merely be aggravated by psychanalysis. Elusive modifications of personality, associated with left-handedness, have been noted in the lives of men of extraordinary ability. Stephen Paget [7] gives a good illustration of this in his life of Sir Victor Horsley: "And he and Gerald had a way of pronouncing th as v. In the use of his hands he was absolutely ambidextrous: he had been left-handed to begin with. He could even draw on wood equally well with either hand. His movements were quick and purposeful . . . There are lives, and his was one of them, at which we are able to warm our hands: the pity is that so many people —it was more his fault than theirs—did not warm their hands but

5. Stier, Ewald: Untersuchungen über Linkshändigkeit und die funktionellen Differenzen der Hirnhälften, Jena, G. Fischer, 1911, p. 302.

6. Translation by the author.

7. Paget, Stephen: Sir Victor Horsley, London, Constable and Co., 1919, pp. 263-265.

burned their fingers at his life." May we not here read a lesson in the life of a man of genius?

CONCLUSIONS

1. It is possible to differentiate established left-handedness from sinistrality by means of performance tests.

2. Both left-handed and sinistral musicians are prone to various forms of muscle incoordination and a defective sense of rhythm.

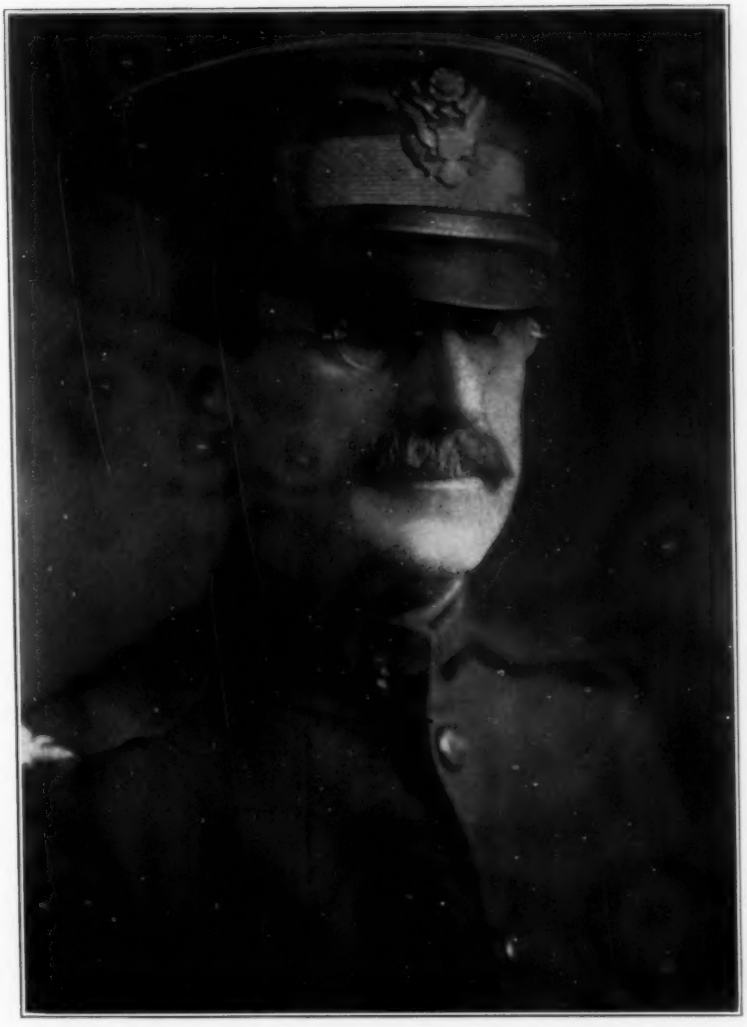
3. The combined values for left-handedness and sinistrality were found to be 8 per cent. in iron-workers, 22 per cent. in relief-home inmates and 32 per cent. in musicians.

4. Left-handedness and sinistrality usually are indicative of the psychopathic constitution.

5. Owing to the imperfect sense of balance which is so often to be noted in sinistrals, it is suggested that what is known as "the feel of the airship,"[8] probably is not as highly developed in sinistral as in dextral pilots.

2216 Steiner Street.

8. Air Service Manual, Washington, D. C., 1919, p. 246.



PEARCE BAILEY, M.D.

1865-1922

Obituary

PEARCE BAILEY, M.D.

In June, 1918, I heard Dr. Pearce Bailey make a report of the War Work Committee of which he was chairman. It told of what he, working with Dr. Salmon and Dr. Paton, had done during the previous year. It was a record of splendid achievements, and I felt that here was a man who had done something wise and notably worth while. It deepened tremendously my respect and friendship for the man. In this year of war work, methods had been outlined for treating and caring for patients with mental and nervous diseases, plans being made for neuropsychopathic hospitals, their equipment, administration and personnel. Later Dr. Bailey studied the methods that were being pursued in Canadian hospitals, and he utilized in his government service the experience there obtained.

A division of neurology and psychiatry was introduced into the army, and for the first time the importance of this kind of service was recognized at Washington. Dr. Bailey was made chief of this new division. Those who know his work there better than I say: "He built a new department and one of vast importance for the efficiency and morale of the army through the elimination of the mentally unstable and unfit. The ideas he effected in this work later were made a part of the psychological work undertaken by the armies of this country's allies."

Dr. Bailey was successful because of his quick and intelligent grasp of problems, because of his administrative skill, and perhaps most of all because it was seen that he was working unselfishly, and not for any personal gain. Entering the service in July, 1917, he was promoted to the rank of lieutenant colonel and finally to colonel, and was discharged from the service in February, 1919, with the Distinguished Service Medal.

He must then have felt that he had developed administrative skill, and his friends knew that he had wisdom, courage, broad vision and initiative. He was therefore urged by some of us to take the position of chairman of the New York State Commission for Mental Defectives, from which Dr. Walter B. James had recently resigned. He accepted the task and he proved again that he possessed the qualities needed for work in this new and difficult field. Governor Miller has given a spontaneous tribute to his efficiency. He said:

"Dr. Bailey was a man who possessed the very rare combination of public spirit, broad vision and practical common sense. And he was

developing a sane and constructive policy in dealing with the problem of caring for and treating mental defectives."

The limited amount of space does not permit me to tell the whole story, but I wish to record that he succeeded for the first time in segregating the defective delinquents and in showing that mental defectives should not be treated with or as criminals any more than should the insane. His commissionership of defectives involved a good deal that was laborious or unpleasant, but he told me after a year of this work that he liked it and was going to continue. His literary skill and imagination helped him in the propaganda of his cause. A short time ago he published an article in a Sunday paper entitled "Shall We Murder the Children?" This was in reference to the fact that two adults of the mental age of 6 years had recently been executed for murder.

But his duties as commissioner took only part of his time, and he proceeded to work out a scheme about which he had long thought and written. This was the problem of the misfit or maladjusted adolescents. He recognized that a marked percentage of maturing lads in the preparatory schools and colleges were not getting on as they should. They were giving trouble to their parents and teachers; they were not, perhaps, bad or defective, but they were not suited to the demands of the ordinary educational schedule. They were dropped or expelled or sent to special schools. No serious and expert study had ever been made of their physical and mental condition or temperamental trends. Dr. Bailey said: "Let us look youth in the face. We will find that among those who are above the level of the moron there are great differences in intelligence and ability. All our boys and girls are not clever. Twenty per cent. of freshmen do not become sophomores; 15 per cent. of sophomores do not become juniors; 10 per cent. of our normal adult population are economic failures." These facts as to the varying capacity of our supposedly normal adolescents led him to organize the Classification Clinic which now exists and functions with a staff of men and women competent to analyze, and to try to solve, the problems of "difficult" children.

So much for the later activities of Dr. Bailey's career. It was during this period that he became one of the editors of the ARCHIVES OF NEUROLOGY AND PSYCHIATRY. He had really found himself, and death cut him down just when he was richly equipped for original and beneficent work.

Dr. Bailey was born in New York, July 12, 1865. He received the degree of Bachelor of Arts at Princeton in 1886 and was graduated in medicine from Columbia University in 1889. He studied abroad, much of the time in France. He was adjunct professor of neurology at Columbia from 1906 to 1910; consulting neurologist at St. Luke's,

Roosevelt, New York, Orthopaedic, Manhattan State and St. John's hospitals. He was a member of the American Medical Association, former president of the American Neurological Association, New York; president of the New York Neurological Society from 1903 to 1905, member of the New York Pathological Society and Academy of Medicine. He was one of the founders of the New York Neurological Institute, in East Sixty-Seventh Street, where he devoted himself to hospital work for many years.

He married Edith Black, of New York, who died about ten years ago. He is survived by two sons and two daughters.

Dr. Bailey early achieved a position of distinction in neurology. His bent was toward neuropathology and organic neurology. But he became interested in traumatic problems and wrote, in 1898, at the urging of Dr. Starr, "Accident and Injury; Their Relations to Disease of the Nervous System." This work was well done. The volume might have been much larger and fuller, but I think Dr. Bailey felt that such works are never final and can never be complete. He gave it the amount of space that was due the topic at that time.

Dr. Bailey wrote many articles on neurologic, psychiatric and allied subjects. His most numerous contributions were to organic neurology and to its traumatic and surgical phases. These articles all have value and contribute to our store of knowledge. I think that his articles touching on neuropsychiatry and social problems are now the more interesting, and they can be read with profit for a long time. "Faith Cures," "Crime and Defectives," "Efficiency as a Medical Problem," "Expert Testimony," "War's Big Lesson in Mental and Nervous Disease," "Neurology and Internal Medicine," were some of his themes. His article on "Voltaire's Relation to Medicine" is an interesting and real contribution to history.

It is an interesting fact that Dr. Bailey did so much important work characterized by originality, initiative and facile receptivity of mind after he was 50 years of age.

Dr. Bailey had an acute intelligence and saw quickly the whole of a problem to the very end. In fact, his quickness of comprehension led him to see that certain things which appeared important were really not worth while; this gave him a reputation for taking up and dropping things very quickly. He was, however, tenacious enough in matters which he saw were fundamentally interesting and worth while.

Dr. Bailey did not have a pedagogic mind, and I think he never cared deeply for teaching. He had a clear, convincing style, however, and could present a topic admirably. He was not a controversialist, but he knew well how to conduct propaganda; instead of arguing, he expounded.

His manner was rather reserved; he was one of the so-called "linear" type—a person with strong emotions, held under good control. Dr. Walter James said that Dr. Bailey was the kind of person one liked better at the second meeting. He possessed humor and wit, and though apparently serious, he was always ready to break into merriment. In spite of his reserve of manner, he had many friends because of his cultural interests, his social qualities and his personal worth, and I doubt that he had enemies. Dr. Bailey had that peculiar quality which enables one to love persons one does not altogether like; "He does terrible things, but I am very fond of him," he would say. He was fair-minded and just in his estimate of men and character.

His talents as a writer of imaginative prose were of a high order; he might have been one of our leading story writers and dramatists. His trend was especially to the dramatic, and his tales ended with a thrill. That at least was the style of literature he gave us at the meetings of the Charaka Club, of which he was a charter member, and in whose volumes are some of his best tales.

Dr Bailey had many friends outside of the medical profession, who recognized his talents and social qualities. His prominence and success in his army work, his activities as commissioner, his new and courageous venture into the adolescent problem, all had conspired to give him unusually wide prominence and importance. The sadness of his domestic life, the loss of his brilliant wife, leaving four young children, had also made his friends feel an especial tenderness toward him.

His death is felt widely and keenly and in a way that I cannot adequately portray. At a meeting of the Charaka Club on February 15, Dr. Peterson offered a resolution which expresses with a personal touch what I would like to say in conclusion.

"Pearce Bailey was one of our founders, one of our best beloved members. During many years his intellect, his wit and his humor have lent a charm to our gatherings which will be sadly missed. We respected and admired him for his integrity of character, his fearless independence of thinking and for the quiet courage with which he bore many sorrows. His fine and striking personality always made itself felt. What he wrote for us was unique in its style, in its keen psychologic analysis and dramatic finish. He has an enduring place in our hearts."

CHARLES L. DANA.

Abstracts from Current Literature

THE RELATION OF THE CEREBROSPINAL FLUID TO THE CIRCULATION OF THE BLOOD AND TO THE NERVOUS ELEMENTS OF THE CEREBROSPINAL AXIS. L. STERN, Schweiz. Arch. f. Neurol. u. Psychiat. **8**:215-232, 1921.

While the cerebrospinal fluid has been made the object of numerous researches, the conclusions reached have not always been in harmony. For example, there is still a great deal of discussion as to the exact course of the cerebrospinal fluid within the nervous structures. According to Goldman, the perivascular space of Robin serves as a lymphatic pathway, communicating on the one hand with the pericellular spaces of Mott and on the other with the subarachnoid cavity. As to the nature of cerebrospinal fluid there is also a difference of opinion, some considering it a secretion of the choroid plexus, others a product of filtration or dialysis of the blood plasma.

The physical properties and chemical analysis of the normal fluid are then reviewed in detail. From the standpoint of chemical analysis alone, it seems unlikely that the cerebrospinal fluid can be considered closely related to the blood plasma. Of all liquids in the animal organism, only the aqueous humor of the eye and the fluid within the ear present any resemblance to the spinal fluid. The analogy becomes closer from both physiologic and teleologic standpoints as these fluids find themselves in immediate contact with nervous elements and in all probability play a similar rôle. The pertinent question arises as to whether this fluid is concerned with the nutrition of the nervous elements.

The writer has long been interested in the permeability of the choroid plexus to various substances. The literature is replete with contributions reporting the passage of diverse substances from the circulation into the cerebrospinal fluid. For the most part, however, these cases have been pathologic and alterations in the vessels and meninges could not be excluded. Experiments on normal animals have been relatively few.

Stern investigated this subject by the following method: In order to prevent the rapid elimination of substances introduced into the general circulation through the kidneys and to increase their concentration within the blood, a double nephrectomy under ether anesthesia was done on dogs, cats, rabbits and guinea-pigs. The substance to be examined was then injected intravenously, under the skin, or into the peritoneal cavity. After varying periods of time the animal was killed by bleeding, and the blood, the cerebrospinal fluid, and the nervous tissues were examined by chemical and biologic methods.

It was found that sodium bromid injected into the general circulation was regularly found in the spinal fluid and in the nervous tissue within seventy-five minutes. The concentration of this salt in the fluid was twice as great as in the nervous tissue and half as great as in the blood. Sodium salicylate, injected in a similar manner, was found in the spinal fluid and in the nervous tissues at the end of an hour. Sodium sulphocyanate was found in less concentration in the spinal fluid than in the blood but in greater concentration than in the nervous tissue. Sodium picrate was found in the spinal fluid and in the nervous structures of all the animals, except in those of the rabbit in which there was remarkable inconstancy. Strychnin could be discovered in the spinal

fluid and in the nervous tissue as early as twenty minutes after injection into the general circulation; the concentration was greater in the nervous structures than in the blood which could be explained only by assuming a definite affinity of this alkaloid for the nervous structures. Morphin was found in the spinal fluid and in the nervous structures at the end of eighteen minutes. Atropin was also found, however, in smaller concentration than in the blood; the results differed in the dog and in the rabbit. Santonin was found in the spinal fluid and in the nervous tissue after intraperitoneal injection. Sodium iodid and sodium ferrocyanid could be discovered neither in the spinal fluid nor in the nervous tissues regardless of the dosage or the method of administration. The same applied to curare. Among the dyes injected, eosin gave a negative result. Fluorescein was not discovered in the spinal fluid, while the choroid plexus was intensely stained and greatly distended. Uranin was not found in the spinal fluid, nervous structures, or even in the choroid plexus; however, intense fluorescence and deep staining were noted in the aqueous humor of the eye on the same side that it was injected into the carotid artery. Methyl violet gave discordant results; it is interesting to note that such substances as sodium iodid and sodium ferrocyanid which normally do not penetrate, when injected simultaneously with methyl violet were regularly found in the spinal fluid and in the nervous tissues whenever the methyl violet penetrated, but were not found in these structures when the dye did not penetrate. According to Veneziani, methyl violet may produce alterations in the choroid plexus leading to the arrest of spinal fluid production. It was impossible to decide whether epinephrin circulating in the blood was prevented from entering the spinal fluid or whether its absence in this liquid was due to its destruction. Bile pigments could not be demonstrated in the spinal fluid either following injection of bile into the general circulation or following ligature of the bile ducts; on the contrary, bile salts were found in the spinal fluid either following injection of bile or ligature of the bile duct. It is possible, therefore, that it is the bile salts rather than the pigments that produce some of the nervous phenomena noted in grave cases of icterus. The search for antibodies in the spinal fluid gave negative results; neither cytolysins, neurotoxins, nor precipitins could be demonstrated in the spinal fluid of immunized animals.

The "hemato-encephalic barrier" is supposed by Stern to functionate in a physiologic rather than an anatomic manner. The exact mechanism is not fully understood. It was a striking fact that whenever a substance introduced into the general circulation was found in the spinal fluid it was also found in the nervous tissues. Furthermore, no substance found in the nervous tissue could not be demonstrated in the spinal fluid. Once they entered the spinal fluid they rapidly found their way into the nervous structures and, if the substance introduced was toxic or irritating, grave symptoms appeared at once. In fact, introduction into the spinal fluid was followed by more certain results than the direct application to nervous structures.

According to the work of Goldmann, the injection of trypan-blue into the subarachnoid space by the vital staining method shows penetration of the brain substance to the nerve cells. The spinal fluid consequently enjoys the same rôle in the metabolism of the brain that the lymph does in that of the cells of the organism. The general course that these substances take would be from the blood into the ventricular spinal fluid, thence through to the ventricular structures into the nervous parenchyma, thence to the subarachnoid spinal fluid, and finally back into the blood.

WOLTMAN, Rochester, Minn.

SCLERODERMIC DYSTROPHIES. HANS CURSCHMANN, M.D., Med. Klin. **17**:1223 (Oct. 9) 1921.

It is Curschmann's belief that the dominating symptom of a dystrophy may be a sclerodermic manifestation. He reports six cases; the first in great detail. A man, aged 42, with a negative family history, was unable to complete school because of mental deficiency. Since puberty, he had had infrequent epileptic attacks. At the age of 19, he developed swelling and tingling of the fingers with pain and gangrene. Gradual thinning of the fingers, induration of the skin and stiffness of the joints developed. Of late years, the gangrene had subsided, the pains had disappeared and the former sweating of the hands had ceased. With the changes in the fingers, painless loss of the teeth occurred. Recently, the patient complained of increasing weakness, loss of libido, constipation, tachycardia and dyspnea.

The hands showed classical sclerodactylia, with evidence of residual Raynaud's disease. The head was small; the nose was long and thin; the skin of the forehead was fine, with many folds. Over the face were many small telangiectases. The hair was thin and the beard was spare. The blood pressure ranged from 85 to 90 systolic, the pulse varied from 60 to 64. The blood picture showed from 9 to 18 per cent. eosinophils. There was no free hydrochloric acid.

The neurologic findings beyond the epilepsy and feeblemindedness were essentially negative. A roentgenogram of the hands revealed atrophy of the distal phalanges.

Skin tests with morphin and epinephrin showed marked delay in reaction, which was mild and transitory.

The patient thus presented a syndrome of sclerodactylia and scleroderma of the face, anhidrosis, loss of teeth, poor sexual development, impotence, pigmentation, adynamia, achlorhydria, continued low blood pressure, eosinophilia, epilepsy, apical tuberculosis and progressive cachexia, which was the most striking of all the symptoms.

Curschmann speaks of this syndrome as a dystrophy with signs of pluriglandular deficiency, in which the scleroderma is a coordinating symptom. He does not consider this an unusual syndrome and he believes that the more we look upon symmetrical scleroderma as a syndrome of glandular function, the more frequently will we see this picture. It is the author's belief, both from his own observation and from the observation of others, that scleroderma is usually associated with a more or less general dystrophy.

The frequency of these "sclerodermic dystrophies" is shown by the occurrence of seven cases in Curschmann's clinic during the past two years.

Five other cases are reported in some detail, the outstanding features being scleroderma, achylia, pigmentation, hypotension, usually eosinophilia and general cachexia. The pluriglandular symptoms vary from case to case; at times there are definite changes in sexual characteristics or these may be absent. Tetany is not an unusual finding, and may be attributed to the parathyroid. One patient had typical tetany attacks in the hands with bronchial asthma attacks.

C. von Noorden described a condition called "degeneratio genitoscleroderma tica" in his work on chlorosis, in which he considered it to be an equivalent of chlorosis. Von Noorden's cases were in previously healthy women who, following an acute infection, ceased menstruating; became weak and cachectic; presented hyperglobulia; indigestion, especially pancreatic;

trophic disturbance of the fingers ("Glanz finger"), etc., and finally developed scleroderma. Pituitary disturbances also might appear.

In this "young girl" type of pluriglandular deficiency, the scleroderma is relatively late and not a striking symptom—a characteristic example of a symptomatic scleroderma. The scleroderma may be entirely absent, the disease process then being called Noorden's disease. Curschmann has described such a case without any scleroderma.

If one considers von Noorden's type and the numerous other forms described in the literature in which the thyroid, pituitary, suprarenal and other endocrine symptoms may occur, with the type of cases described by the author, it is Curschmann's belief that the use of a general term, such as sclerodermic dystrophies, is more applicable than the attempt to establish a single glandular agent as the causative factor in some special case.

In attempting to determine the etiology of this sclerodermic change, it seems self-evident that no single general cause can be held responsible. It is doubtful that it is the result of a pluriglandular change; it seems more rational to attempt to find a cause for both the pluriglandular change and the scleroderma as they seem coordinated. This search would lead to the sympathetic or parasympathetic system of both cerebral and spinal origin. Just how the sympathetic system is altered, whether by toxins or infections, remains a question.

The assumption that the pluriglandular changes and the scleroderma are coordinated results of a sympathetic disturbance is of special interest because of late many workers are attributing to the skin endocrine functions. These properties of the skin, as in other dystrophies with pluriglandular syndromes, in vasomotor and trophic neuroses, etc., present a new problem which will require further investigation.

MOERSCH, Rochester, Minn.

EUGENICS AS A FACTOR IN THE PREVENTION OF MENTAL DISEASE. HORATIO M. POLLOCK, State Hosp. Quart. **7**: No. 1 (Nov.) 1921.

Inheritance of the neuropathic constitution may be taken for granted. Much evidence has been adduced to prove that such inheritance occurs in accordance with mendelian laws, but the subject is so complicated that more comprehensive studies need to be made before we may consider the matter as settled. The application of skilfully devised measures of intelligence has shown us that there are many grades of intelligence between the idiot and the superaverage. The so-called normal persons represent many types, the extremes of which are as far apart as the moron is from the low-grade normal person. Recent studies of temperamental abnormalities have also revealed a wide variety of types and combinations. These abnormalities or marked peculiarities seem to be more or less dissociated from intellectual capacity. Children with superaverage intelligence are frequently seclusive and morons often seem to be temperamentally normal. It becomes difficult, therefore, to establish standards of normality and to draw fixed lines between the normal and the neuropathic state. This is especially true in studying family histories when judgment must be based on reports of untrained observers.

The data collected by the New York State Hospital Commission relative to family history seems to indicate that slightly more than half of the ascertained cases have no discoverable hereditary basis. If more thorough inquiries were made the proportion of patients with an unfavorable family history might

be increased, but the significance of the history in relation to the family stock is open to question in many cases.

In New York state hospitals for some years past have been studied both the intellectual and temperamental makeup of patients admitted for the first time. In 1920, it was found that of the ascertained cases 61 per cent. were temperamentally normal and 88 per cent. were rated as intellectually normal. Only about 7 per cent. of the patients were both temperamentally and intellectually abnormal. The proportion of patients with an abnormal makeup varied considerably in the different groups of psychoses. For example, in the dementia praecox group in 1920, 61 per cent. were rated as temperamentally abnormal while in the manic-depressive group only 33 per cent. were so rated.

The absence of marked abnormalities prior to the onset of the psychosis cannot be construed as conclusive evidence that there are no hereditary defects in the makeup, neither can the development of the psychosis be taken as proof of a defective constitution. All the facts in connection with the onset of the mental disorder and previous reactions must be brought together before the constitutional makeup of the patient can be positively determined.

Notwithstanding these and many other complications, there is abundant evidence that mental disorders occur much more frequently in some family stocks than in others and that prolonged inbreeding of degenerate stocks is productive of most disastrous results.

With the limited knowledge at hand, what is to be done to lessen the burdens imposed on society by the prevalence of mental disease?

Three lines of action are suggested:

1. Environmental stresses may be lessened and natural resistance strengthened.

2. Procreation of defective stock may be checked.

3. Procreation of normal stock may be increased.

Economic and social stresses should be lightened for those unable to withstand them. It is far easier to relieve an overburdened man by taking part of his load than to wait until he is exhausted and then carry him together with his burden. Physicians, parents and teachers should be alert to detect signs of mental disorder and apply the proper remedy before complete breakdown occurs.

Segregation of the mentally defective and epileptic is the prevailing method of limiting procreation among these classes. Its eugenic value is beyond question, but the enormous cost limits its application. As a rule, mental defectives and epileptics cared for in institutions are of low grade. These, if left at liberty, would multiply far less than those of higher grade. Much is to be hoped from the colony plan of segregating mental defectives, as colonies care for high grade defectives and under wise management become self-supporting and may be increased without limit.

A new departure has been made by the state of New York in establishing a separate institution for defective delinquents at Napanoch. This troublesome group has been a serious problem in the jails and prisons of the state and heretofore there has been no satisfactory way of dealing with them. Their segregation should have great eugenic significance.

Something can be done to lessen reproduction among the unfit by enlightened public sentiment and by better marriage laws. Marriage of persons with marked intellectual or temperamental abnormalities should be prohibited.

To prevent the marriage of normal persons with those carrying a neuropathic taint more knowledge of family stocks must be made available. At the present time genealogic records of the average family are wofully meager

and comparatively few are available for public inspection. If we are to improve the race by better marriages genealogic or eugenic bureaus must be established in cities and villages. Data concerning family stocks should show the defects as well as the excellencies and achievements of the individuals recorded and be available to interested parties.

BAILEY, New York.

REPORT OF A CASE OF OPTIC APHASIA AS A CONTRIBUTION TO THE MECHANISM OF SPEECH DISTURBANCES. HEINZ FENDEL, Berl. klin. Wchnschr. No. 38, 1134 (Sept. 19) 1921.

The author reports in detail a case of optic aphasia and illustrates his statements by the use of a diagram taken from Liepmann.

Little advance has been made in recent years in the study of speech mechanism, and Fendel believes that by carefully studied clinical cases much can be hoped for. Anatomic experimentation has proved to be practically useless. The psychologists who have attempted to compare periods of speech development with types of aphasia have found what seem to be insurmountable barriers.

Injury to Wernicke's center or disturbance in association fibers with this center are, in the opinion of the author, the bases for all aphasic disturbances.

The case reported is one of optic aphasia, associated with a right homonymous hemianopsia and pure verbal alexia, with no sensory aphasic disturbances. The patient was a woman, aged 45, who on Feb. 18, 1921, was brought to the hospital in a stupor following a severe erysipelas. When aroused a monophasia was distinguished; she always replied in a senseless manner "da-da-da-da." Examination when feasible showed a peripheral arteriosclerosis; the systolic blood pressure was 160. Urine, blood and spinal fluid were normal. The neurologic examination was negative except for right homonymous hemianopsia. The fundi were negative. A preliminary diagnosis of thrombotic softening in the left occipital region, arteriosclerotic in nature, was made.

Examination of the speech mechanism showed that understanding of speech was intact. Motor commands were executed promptly, repetition unimpaired and spontaneous speech normal. The patient was unable to name recognized objects. When shown a key, she gave evidence of recognizing the object but could not recall its name. When asked, "With what do you lock the door?" the word key was given at once. In this case tactile sense did not help to arouse the visual word image, but auditory sense frequently did. Thus, although she could not name a watch, she immediately recalled the name of the object when it was placed to the ear; this was also true of other objects. With considerable practice the patient regained some ability to name commonplace objects.

The patient was unable to read, could name single letters and by spelling was able to name words. She could not appreciate written commands. Isolated figures were read correctly, and she was able to write and print but with numerous mistakes. Dictation writing was much better but also showed mistakes. Single letters could be copied, but the patient was unable to write names of objects or words without errors.

During the two months of observation the only improvement noted was the patient's ability to name everyday objects. In attempting to explain this speech disturbance the author assumes the presence of a lesion in the posterior portion of the temporal lobe. All cases of optic aphasia at necropsy have

shown a lesion in this general position where the auditory association fibers and the optic auditory commissural tracts converge.

Optic aphasia is truly an optic-auditory conductive aphasia, the centers themselves remaining intact. Oppenheim has seen only one case of word deafness.

The early monophasia in this patient is attributed by the author to the apoplectic affection which extended beyond the borders of the anatomic lesion.

In attempting to determine whether the loss of object recognition and loss of reading is due to disturbance of one or both optic centers, Fendel states that Déjerine and Ferrier assume that only the left center is able to recognize letters. In this case the left center is cut off from the periphery and to arouse optic memories there must be some connection with the right center. If it is assumed that the commissural tract between the visual centers is intact, then also the association tract between the visual center and Wernicke's area on the left side must be intact. Fendel assumes that the connection of the right visual center and the left Wernicke zone was left intact and that by this means the patient was able to accomplish the reading of single letters, but was unable to read complicated words or name objects.

The pure verbal alexia in this instance was not a disturbance *sui generis*, but a mild total alexia, or it might be called a dyslexia. The right hemianopsia was not followed by loss of color sense; therefore it seems to the author that the right hemisphere is not only for object recognition and color recognition, but also for certain object quality recognition.

The author admits that much which has been propounded is theoretical. Numerous other queries are made, and he attempts to answer them. He admits, however, the difficulties that are encountered in any explanation and ventures the hope that more intensive work may help to elucidate some of these interesting phenomena.

MOERSCH, Rochester, Minn.

A PRESENILE DISEASE RESEMBLING PSEUDOSCLEROSIS. CONSTANTIN ECONOMO and PAUL SCHILDER, Ztschr. f. d. ges. Neurol. u. Psychiat. **55**:1, 1920.

The authors relate the case of a man whose illness began in the fiftieth year and continued with remissions for five years, when he finally died in a remission lasting five months. Clinically the picture was one of increasing muscular rigidity with occasional periods of mental confusion. His last attack was marked by staring face and eyes, difficulty of movements of eyeballs outward and downward, half opened mouth, dysarthria and explosive speech, dysphagia and rigid lower extremities in extension which were adducted and internally rotated. The tendon reflexes although masked by rigidity were present. There were no pathologic pyramidal tract reflexes. In the strict sense, paralysis or paresis was absent. Neither was somatic sensation affected.

The rigidity was characterized by the following special features: There was an appreciable latent interval before voluntary effort could initiate movement. When such movement was established it would be arrested by a recurrence of the rigidity which affected agonists and antagonists alike. Passive motion, if made gently, would tend to relax the rigidity; if brusquely, would tend to increase it. As in a normal case, resistance applied against movement in an extremity would be followed by the rebound phenomenon when such resistance was suddenly released. One rebound would be followed by another, the extremity going through a sort of pendulum movement. Tendency to asso-

ciated movements was expressed by an increase of rigidity in the general musculature when the rigidity was increased in one extremity. At rest form tonus was increased.

The exitus was accompanied by tonic extension of the upper extremities. At necropsy an acute bronchitis was found and a cardiovascular nephritic degeneration of mild degree. The brain showed a slight chronic internal hydrocephalus. Macroscopically the liver was negative. The histopathologic examination of the nervous system revealed the following lesions:

Cerebral Cortex: Fat in the ganglion cells was increased, and also the number of satellite cells, especially in the deeper layers of the cortex. These changes were not marked, but they exceeded the changes normally found at the age of the patient.

Cerebral White Matter: The small glial elements and degeneration products about the vessels were increased.

Basal Ganglions: In the basal portion of the corpus striatum and neighboring parts of the substantia innominata there occurred a diffuse glia cell increase of small lymphocyte-like cells rich in chromatin and revealing no protoplasmic ring with the methyl green—pyronin stain. In addition there was an increase in glial fibers. The globus pallidus showed large and small accumulations of degeneration products in the media, about the endothelium of the capillaries and also free in the surrounding tissues. It was determined that these masses contained both fat and protein.

Cerebellum: The most noteworthy finding was a high grade intracellular fatty deposit in the molecular layer of the cortex.

The spinal cord was not examined.

The liver showed a moderate interstitial hepatitis.

Preliminary to a discussion of the classification of this case, which they believe to be a distinct affection, the authors review the literature of pseudosclerosis and allied clinical forms, including Wilson's disease. The criticism is made that in Wilson's article an insufficient analysis and characterization of hypertonus is made. Sherrington's classical experiments of decerebrate rigidity are reviewed and application made to the forms under discussion. The authors accept as a fact that striatum lesions produce hypertonus, admitting, however, that in certain cases the mechanism may be produced by cerebral decompensation. Two cases reported by Woerkom, occurring in the praesenium without a heredodegenerative or constitutional basis, are most closely related to the personal case of the authors. The closely related entities of paralysis agitans and atrophia-olivo-pontocerebellaris are discussed from a differential standpoint.

Anatomically in the reported case there are certain distinguishing features. The glial increase in the corpus striatum and the characteristic small-celled glia are contrasted with the large-celled glia found in pseudosclerosis. From Wilson's disease it differs by the absence of any disintegration of the lenticular nucleus. The cerebellar changes are without analogy. Nosologically the case is also distinguished from pseudosclerosis and Wilson's disease.

SCHALLER, San Francisco.

BROMID DELIRIUM. BEN KARPMAN, State Hosp. Quart. **7**:66 (Nov.) 1921.

The symptoms of bromid delirium have a varied psychotic picture which has much in common with the pictures of many psychoses. The marked intellectual disturbances with secondary affective deterioration, incapacity for complicated

thinking, the defective and incoherent speech, diffuse defects in memory and retention with consequent disordered understanding of the environment, appear as evidences of diffuse cortical degeneration and thus bear a strong family resemblance to organic psychoses, such as general paralysis or arteriosclerotic dementia; although, as suggested by Hoch, in delirium there is more of a weakness than an actual loss and the disorder is transient, acute and recoverable. At the same time the undoubted presence of pressure activity, emotional instability, alternating excitement and depression, destructiveness, distractibility, and flight of ideas suggest strongly an underlying affective mechanism that closely resembles the manic-depressive psychoses. On the other hand, there stand out dissociation from reality, hallucinations and delusions, dream-like experiences, stuporous states, intellectual impairment, manifest disharmony between the intellectual content and its affective expression, bringing it close to the trend reactions such as we observe in the dementia praecox group. It is thus clear that the presence in delirium of a mixture of organic, affective and trend reactions precludes the possibility of grouping it with any one of these types of reaction. But we may approach the subject in another way. We may classify the deliria not on the basis of symptoms and reaction, but on the basis of etiology and content. We may group certain deliria showing affective or trend reactions and call these psychogenic in contrast to the organic deliria for which we can claim a physical basis; meaning by psychogenic such disturbances as are clearly the result of, and lie properly within the field of, mental activity and could not be conceived as arising otherwise. It is, however, an inadequate and poorly directed mental activity; the patient finds himself face to face with an unbearable situation to which he cannot or will not adjust himself, and instead of trying to solve the problem, he runs away from it and develops a psychosis, the outstanding feature of which is the unconscious and symbolic striving for the realization of the ungratified wish. But we know that in many toxic deliria also symbolic representations of wish fulfilments are frequently observed; hence, we cannot separate the psychogenic from organic deliria even on the basis of content, for although the latter may vary in strength, these deliria are obviously the same in character. It thus appears that infectious and toxic agents in organic deliria and mental causes in psychogenic deliria produce their effect in much the same way—through intoxication and exhaustion. In psychogenic delirium mental causes have brought about the exhaustion and clearly dominate the entire picture of the psychotic episode, which would not have taken place were it not primarily for the mental causes; in other words, the psychogenic factor is a disease symptom as well as an etiologic agent, whereas in toxic delirium the toxemia simply awakens the deep seated conflicts and strivings that have heretofore been lying dormant. But if we bear in mind that even in deliria with a toxic etiology, the mental make-up and attitude of the patient have, to begin with, determined the susceptibility to intoxication as well as later determining the manifest expression of it, it will become obvious that the toxic agent has entered here merely as one of the factors in the etiologic constellation and that neither on the basis of reaction or symptoms, nor on the basis of etiology and content is there an inherent justification in separating deliria into psychogenic and organic. They are all essentially the same in nature and content, they all have the same nucleus—the specific delirious alteration of personality which is characteristic of the particular person and is symptomatic of the difficulties under which he lived.

But whether or not we separate delirium into groups and types, the diagnosis of delirium is, as a rule, not difficult. The differential diagnosis, how-

ever, is not only difficult but often quite impossible for the reasons given; yet it is obviously important since the prognosis depends essentially on the etiology. Specifically, in our instance we find that no clear cut differentiation can be drawn in bromid delirium from other deliria and the differential points offered here are merely suggestive.

From delirium occurring in the course of infectious diseases, the bromid delirium may be largely differentiated by the lack of febrile and infection symptoms, and by the eruption usually present. An intense toxemia, with the patient lying in bed in a condition of lethargy and stupor, carphologia, low muttering delirium bordering on coma, gives us a clue to a typhoid type of delirium.

From exogenous psychoses such as develop as a result of using chloral, cannabis indica, paraldehyd and other drugs, the differentiation is quite impossible, since they all present practically the same clinical picture.

POLLOCK, Albany, N. Y.

A CONTRIBUTION ON THE PATHOLOGIC ANATOMY OF POST-HEMIPLEGIC ATHETOSIS. H. STECK, Schweizer Arch. f. Neurol. u Psychiat. **8**:75-85, 1921.

Recently several contributions have appeared which concern the so-called extrapyramidal, or striate, disturbances of motility. The following report is based on the examination of a case reported by von Monakow in the second edition of "Gehirnpathologie," as a typical example of pure posthemiplegic athetosis, which he had the opportunity of following for twenty years:

Mrs. K. B., aged 67 years, with a negative family history, was perfectly well until the age of 27, when she suffered a left-sided stroke, following which there was residual weakness of the left arm and leg. At the age of 44, she suffered a left-sided hemiplegic stroke with loss of consciousness and some disturbance of speech. Almost complete improvement followed. At the age of 64, she had a third stroke, with partial recovery. Abnormal movements were said to have followed the first attack; these disappeared later. However, intense pain, in the left half of the body, particularly in the arm and shoulder, persisted. After the second stroke, involuntary, slow, irregular movements appeared in the left arm and fingers, and in the left leg and foot, which, except during sleep, continued uninterruptedly. There was a mild paresis of the muscles of the left side of the mouth; the left leg was paretic and somewhat abducted, with increased muscular tonicity; there was dorsal flexion of the left great toe. It was quite impossible for the patient to arrest these characteristically athetoid movements, even momentarily. There was a slight diffuse atrophy of the left arm and leg. No demonstrable loss of sensibility, save slight disturbance of stereognosis, was present. The patient was able, at times, to move some of the muscles voluntarily; however, this had to be done at a time when that particular muscle was not involved in the athetotic movement.

Necropsy revealed some interesting findings. There was a mitral endocarditis, and these changes in the nervous system: The dura showed no change on its external surface; but on its internal surface, over the left side, it was covered with a thin layer of brownish blood pigment. There was marked edema of the pia. The gyri were comparatively small, the sulci broad; however, there was no noteworthy asymmetry in comparing the two halves. The contour of the left lateral ventricle was normal; but on the floor of the right ventricle could be seen several gray-white depressions, one of which was

present over the head of the caudate nucleus, and a second over the latero-anterior portion of the optic thalamus. On incision a smooth walled cyst, about 1 cm. in diameter, was found in the head of the caudate nucleus and another, 2.5 cm. in diameter, in the left lenticular nucleus and impinging on the middle and posterior portion of the internal capsule and of the optic thalamus. The arteries of the base were delicate and without pathologic changes. Unfortunately, only the right half of the brain stem was preserved; the anterior portion was fixed in Mueller's fluid, embedded in celloidin, and frontal sections were made, which were stained by the Weigert-Pal method and with carmin. The posterior half, containing the nucleus ruber, was stained with toluidin-blue and carmin.

Microscopic examination demonstrated that both cysts communicated through a rent in the small intervening membrane. The smaller of the two cysts was located mainly in the caudate nucleus, while the ventral one lay entirely within the putamen, impinging on the internal capsule. The walls of the cysts were of a brownish-red color, from 1 to 2 mm. thick, and contained no nerve cells, but showed a slight increase in glia cells. In the remaining portions of the caudate nucleus and the putamen, an occasional nerve cell could be found. The myelinated fibers passing through the putamen were diminished in number and in their stead there was an increase in glia cells. Shrinkage, tigrolysis and satellitosis of the remaining nerve cells bespoke an early degeneration. In the posterior portion of the globus pallidus could be noted a definite rarefaction with disappearance of some of the larger nerve cells; the anterior part was normal. Secondary degeneration was evident in the lenticulocaudate fibers passing through the internal capsule. The pyramidal tracts, however, so far as examination permitted, were nowhere diminished in volume. The corpus Luysi was normal, as were the substantia perforata and the red nucleus. The thalamus showed some cellular disintegration of the lateral and ventral but not in the medial nuclei. In brief, there was extensive degeneration of the body of the caudate nucleus, partial destruction of the anterior portion, complete destruction of the middle, and partial destruction of the posterior third of the putamen and some disturbance of the lenticulostriate portion of the internal capsule; the medial portion of the thalamus, the red nucleus, the substantia perforata anterior, the locus niger, the peduncles and the pons were normal.

The finding of note in this case is the complete preservation of the red nucleus and the apparent integrity of the corticopallidal fibers described by Déjerine. The correlation of cases reported in the literature is difficult. It is well known that there are cases reported with lesions in these portions of the brain that are not associated with athetosis. In fact, of eleven patients with lesions in these areas in which Steck had the opportunity of conducting a necropsy, only one had athetotis, three had tremor, a fourth rigidity; the remainder were normal. This would indicate that disturbance of the striate body can result in very different pictures.

In comparing cases of posthemiplegic chorea and athetosis, which have been reported clinically and pathologically, a difference in localization of lesions becomes apparent. Cases of hemiplegic athetosis resemble those of athetosis double in their relation to lesions of the striate body, while cases of posthemiplegic chorea show no striate lesion, but show a lesion of the brachium conjunctivum. A detailed discussion of the clinical pictures and the underlying anatomic findings would, at present, be premature and unconvincing.

WOLTMAN, Rochester, Minn.

THE CEREBROSPINAL FLUID IN SYPHILITIC PSYCHIC DISTURBANCES. SIGURD DAHLSTRÖM and SOFUS WIDERØE, Ztschr. f. d. ges. Neurol. u. Psychiat. **72**:75 (Oct.) 1921.

This is an important contribution to the problem of the communication of the spinal fluid in the different loci of the cerebrospinal fluid system. The authors call attention to the fact that in syphilitic diseases of the central nervous system it is not unusual to find that the serologic, cytologic and chemical constituents of the fluid from the ventricle and lumbar sacs differ. Thus in a group of seven cases they indicate the difference as shown by the Wassermann reaction, cell count, albumin and globulin reactions in these two loci. They endeavor to determine whether there is any communication between these two areas. For this purpose they have used two methods: one, the injection of neutral phenolsulphonephthalein in the lumbar sac and also in the brain ventricle. The second method is the injection of air after the technic of Dandy. One half hour after the intraventricular injection of 1 c.c. of phenolsulphonephthalein they were able to regain it from the spinal fluid in the six cases in which it was attempted. The more fluid that was withdrawn from the lumbar sac, the redder it became. In one case 1 c.c. of phenolsulphonephthalein was injected into the lumbar sac. About five minutes later a ventricular puncture was made and 15 c.c. of fluid were withdrawn in three tubes, 5 c.c. to each tube. The first tube was weakly colored, the second more strongly colored, and the third showed the most dye. They concluded from this that passage from the lumbar sac upward is possible. Two hours after the injection of phenolsulphonephthalein into the cerebrospinal system, the bladders in all six of the patients were catheterized and the urine tested for the dye. In two cases no dye was obtained, in three, from 2 to 3 per cent. only, and in the sixth, 8 per cent. was obtained. They call attention to the small amount that came into the urine in these syphilitic cases, being much less than the quantity Dandy has given as normal. They also injected air, according to the method of Dandy, into the ventricles and into the lumbar sac at different times. As regards the lumbar injection, they record and show roentgenograms made at the time to prove that the air was easily pushed up into the cisterna magna and lateral ventricles to the cisterna chiasmatica and also over the surface of the brain in what must have been the arachnoid space.

SOLOMON, Boston.

DEVELOPMENT AND REACTION OF THE PLANTAR REFLEX UP TO THE AGE OF TWO YEARS IN BABIES BORN BEFORE TERM. H. BERSOT, Schweizer Arch. f. Neurol. u. Psychiat. **8**:46-74, 1921.

The previous section of this article was abstracted in these columns. Bersot reviews many of the points that he made previously and lays emphasis on the fact that the two fundamental characteristics of the plantar reflex, as seen in early life, are the variability of muscular responses in the feet themselves and the production of associated movements. Among the reactions, extension of the great toe was relatively more constant than movement of the interosseous and lumbricales muscles. The extreme variability of the movements, both on the same side and on the opposite side at various ages, are recorded in tables presented in a manner that does not lend itself to abstracting.

In commenting on the plantar reflex, he states that the general irritability increases for two or three months, then diminishes. The type of reaction also differs, so far as the early movements are slow and of long duration; and

particularly in babies born before term, there is a general tendency to approach the intra-uterine attitude. Later these reactions become more rapid and the semipermanent contractions become less frequent.

Myelinization of the peripheral nerves appears in the fetus of from about 4 to 5 months, which corresponds to the onset of quickening. By the eighth month myelinization is almost complete. In the fifth and sixth month of intra-uterine life, myelinization makes its appearance in the anterior columns, later in the lateral columns. By the ninth or tenth month after birth, the pyramidal tracts have been formed.

Held made the interesting observation that the eye of a dog which is exposed to a bright light, while the opposite eye is closed, is myelinated more rapidly. This suggests an explanation for the variability in response to stimulation of the peripheral nerves.

From the work of Soltmann and Westphal, it appears that the sensory and motor nerves are less irritable in early life than in adult life.

Babinski and his followers adhere to the point of view that an extension of the great toe invariably means an anatomic lesion of the nervous system. All writers, however, do not agree with him fully. Attempts have been made to distinguish extension of the great toe from medullary automatism and the defensive reactions. While the various points of view have been ably defended by some and gravely questioned by others, Bersot retains his equanimity by taking side with none of them. He states that variations are to be expected, that the various types of reflexes noted in infants of different stages of development may remain latent and come to light only under certain pathologic conditions.

WOLTMAN, Rochester, Minn.

TREATMENT OF ACUTE POLIOMYELITIS WITH IMMUNE HORSE SERUM. E. C. ROSENOW, J. A. M. A. **77**:588 (Aug. 20) 1921.

In a series of experimental investigations, Rosenow found that monkeys were not protected against intracerebral inoculation of the virus of poliomyelitis by intraspinal injections of immune horse serum. But, on the other hand, he found that intravenous injections did confer protection. Of 259 cases in man treated intravenously and intramuscularly, none of the sixty patients treated in the preparalytic stage died, and no residual paralyses were found. Of sixty-one patients with slight paralysis all but one recovered completely. Out of 123 cases eighteen patients died with advanced paralysis, eleven of them having symptoms of medullary involvement; thirty were left with residual paralysis ,and sixty-one recovered completely. Fifteen cases of sporadic poliomyelitis were treated, with one death and six recoveries with residual paralysis. Of the 259 cases, there was a mortality of 7.3 per cent., made up mostly of those cases in which serum was given late (eleven of the nineteen cases). Residual paralysis was present in 21 per cent., omitting the late results which were not known in fourteen cases. Of 197 paralyzed patients, nineteen (9.6 per cent.) died, which gives a mortality of 4 per cent. by eliminating eleven patients whose cases were hopeless at the time of serum treatment. Twenty-one per cent. of 176 paralyzed patients who survived had residual paralyses.

The efficacy of the serum injection was proportionate to the earliness of the injection, and invariably each successive injection further hastened recovery. The author concludes that immune horse serum has a definite curative value and that an early diagnosis is essential. He recommends spinal puncture in all suspicious cases.

PATTEN, Philadelphia.

THE CIRCULATION OF THE CEREBROSPINAL FLUID. C. von Monakow, Schweiz. Arch. f. Neurol. u. Psychiat. **8**:232-234, 1921.

Monakow, in commenting on Stern's contribution, emphasizes the fact that the work of the latter quite agrees with that undertaken by the author and Kitabayashi on the pathology of the choroid plexus. Doubtless the production of ventricular fluid by the choroid plexus is under control of the hemispheres, judging from the wealth of nonmedulated sympathetic nerve fibers in the plexus. The fluid from the choroid plexus probably enters the ventricular wall through the intercellular ependymal spaces, after which it is readily distributed in all directions along the glia network, nerve fibers and blood vessels, thus delivering substances to structures in a way that could hardly be accomplished by the blood vessels alone. Doubtless the ventricular fluid bathes the parenchyma directly. Waste products of metabolism are then picked up at the periphery of the cortex, leaving by means of the perivascular lymph spaces of His which open directly into the subarachnoid space. Part of it leaves by way of the venous plexus through the sinuses, part through the pacchionian granulations, part accumulates in the larger cisterns, and part finally reaches the lumbar subdural space where it reenters the blood stream.

According to injection experiments of Sicard and others, it appears that insoluble particles are carried off by "Abbauzellen" in the Virchow-Robin spaces from which they enter the meningeal lymph spaces, finally ending in the lymphatics of the neck. There are, accordingly, two outlets from the parenchyma of the brain into the circulation of the blood, one for soluble substances along the path for fluids, the other for suspended and insoluble material largely by means of the cells.

Woltman, Rochester, Minn.

EPIDEMIC HICCUP WITH GENERALIZED MYOCLONIC MOVEMENTS. Ducamp, Carrien, Blonguier de Claret and Tzélépoglon. Bull. de l'Acad. de méd. **86**:249 (Nov. 8) 1921.

A case is reported of a young woman who, in January, 1921, died after a three weeks' febrile illness in which the symptoms were intense hiccup with myoclonus of the legs and abdominal walls not accompanied by disturbances of the ocular musculature either intrinsic or extrinsic or by lethargy. Breathing was gravely disturbed, and this disturbance was the cause of death. Lumbar puncture had shown a normal spinal fluid.

Histologic study showed that at the peduncles, the pial vessels were overcrowded with red cells and presented a marked infiltration of their walls with leukocytes. Toward the center of each peduncle, there was a round cell infiltration and a neurogliar proliferation. The nerve cells showed an incontestable neuronophagia. The locus niger was slightly affected. In the bulbar part of the fourth ventricle the neurogliar proliferation and neuronophagia were more extended than in the peduncles. At the vagal nuclei there was perinuclear chromatolysis and sudan stain demonstrated marked lipoid granulations in the perinuclear region.

Throughout the cervical cord there was congestion together with ependymal round cell infiltration, and in the anterior horns the cells showed widespread chromatolysis.

The authors present their case and give the histologic findings without saying whether in their opinion these findings warrant the inclusion of the case in the epidemic encephalitis group.

Davis, New York.

THE INTERPRETATION OF SEROLOGICAL FINDINGS. STERNE MORSE, State Hosp. Quart. **7**:20 (Nov.) 1921.

In the interpretation of serologic findings which diverge among themselves and remain divergent after repetition one has several possibilities to consider, of which the following are examples:

1. A positive cell count, a negative Wassermann reaction in the spinal fluid, a positive blood Wassermann reaction. In this situation syphilis of the central nervous system is probable unless some other condition, such as epidemic encephalitis or brain tumor, is associated with systemic syphilis.

2. A positive cell count, a faintly positive Wassermann reaction in the spinal fluid (1 or 2 plus in the 0.5 c.c. dilution), and a negative blood Wassermann reaction. Syphilis of the central nervous system is probable.

3. A doubtful cell count (4 to 9 cells), a faintly positive Wassermann reaction in the spinal fluid (1 or 2 plus in the 0.5 c.c. dilution), a strongly positive blood Wassermann reaction. Here also syphilis of the central nervous system is probable.

4. A negative cell count, a positive globulin reaction, a faintly positive Wassermann reaction in the spinal fluid and a positive blood Wassermann reaction. In this group syphilis of the central nervous system may or may not be present, as such findings can apparently occur in systemic syphilis.

POLLOCK, Albany, N. Y.

SPINA BIFIDA. F. C. PYBUS, Lancet **2**:599 (Sept. 17) 1921.

Pybus discusses briefly the following types of spina bifida: spina bifida occulta, meningocele, meningomyelocele, syringomyelocele, and myelocele. Their diagnosis, the congenital malformations accompanying these tumors, the selection of cases for surgical treatment, and the method of operation are also considered.

The author refers to four children in one family who had some degree of spinal defect. The eldest child died at the age of 18 months as a result of spina bifida, type unknown, and hydrocephalus. The second child, aged 10 years, had a spina bifida occulta, indicated by a scar and depression in the upper dorsal region and by slight scoliosis. A roentgenogram showed a defect of the vertebrae in this region. The third child, aged 9 years, had a meningomyelocele in the sacral region. This was treated surgically with excellent results and the patient is now quite well and without deformity, the only residual symptom being an inability to retain urine as long as usual. The only defect in the fourth child is an exaggerated postanal dimple.

POTTER, Mercer, Pa.

NOTE ON THE TECHNIC OF THE TREATMENT OF SYPHILIS. THEODORE D. REED, State Hosp. Quart. **7**:30 (Nov.) 1921.

1. It is desirable in the intravenous administration of neo-arsphenamin, particularly to the insane, who may be disturbed, to administer the drug as rapidly as is compatible with good results.

2. No unfavorable reactions have been noted by us following rapid administrations.

3. No unfavorable reactions, either symptomatically or in the blood, have been noticed after giving neo-arsphenamin in high concentration, that is, 0.9 gm. per 2 or 3 c.c. of double-distilled water.

POLLOCK, Albany, N. Y.

Society Transactions

NEW YORK NEUROLOGICAL SOCIETY

Joint Meeting with the Section of Neurology of the Academy of Medicine, Nov. 1, 1921

FOSTER KENNEDY, M.D., and S. P. GOODHART, M.D., *Presiding Jointly*

A CASE OF FOURTH VENTRICLE TUMOR WITH SURGICAL INTERVENTION. DR. I. S. WECHSLER.

A young housewife, aged 29, was first seen May 25, 1921. She complained that, beginning suddenly in November, 1920, she suffered from dizziness and spells of momentary darkness. The attacks lasted a few seconds; she felt like falling and occasionally did fall, though to no particular side. The dizziness was especially apt to come on when she got up on a chair. To overcome the vertigo she had to hold her head in a rigid position. The spells would disappear for a few weeks. She did not complain of headache, vomiting or tinnitus. She had gained 30 pounds (13.6 kg.) in weight and her menses had become irregular, prolonged and profuse. The gait was normal.

The neurologic findings were practically negative, except for bilateral choked disk of 5 diopters, a somewhat acromegalic face and inequality of pupils. The diagnosis made was tumor of the fourth ventricle—tumor because of the choked disk, and fourth ventricle because of remissions, of the need of holding the head rigid to overcome the vertigo (interpreted as a modified Bruns symptom), and because of the absence of other localizing signs. Tumor of the quadrigeminals was ruled out because of the absence of pupillary signs and of ocular palsies; tumor of the pituitary was also thought unlikely, despite the acromegalic face, gain in weight and menstrual disturbances. Cysticercus was considered.

The patient was admitted to the Mt. Sinai Hospital. The blood Wassermann and urine were negative and the blood chemistry was normal. Eosinophilia of 7 per cent. indicated cysticercus. The roentgen ray showed a normal sella and no signs of pressure. Dr. Elsberg performed a suboccipital craniotomy and found a tumor filling the fourth ventricle. It was adherent to the floor and sides and to the calamus scriptorius. An attempt to remove a piece of the tumor resulted in bleeding, which necessitated a rapid retreat. The tumor appeared to be either a glioma or ependymoma.

Soon she developed some dysarthria, a little staggering, vomiting and slight optic atrophy. At no time did she have nystagmus, pyramidal tract signs, cranial nerve palsies, sensory disturbances, vomiting, headache, rigidity of the neck, polyuria, glycosuria or vagal symptoms. The patient is still living, five months after the operation, and has improved somewhat.

DISCUSSION

DR. M. NEUSTAEDTER: What was the pulse rate?

DR. S. P. GOODHART: Was it practical to make an effort to remove any part of the growth? It seems remarkable indeed that a tumor of such size should have been within the fourth ventricle and given practically no signs. I presume it is because the tumor is soft in character causing only slight

mechanical displacement. It is surprising that a growth so far back should have caused 5 diopters of choked disk and no indication of involvement of adjacent structures.

DR. B. ONUF: Was one of the symptoms titubation of gait?

DR. WECHSLER: The pulse was always within normal limits and so was the respiration, except during and immediately following the operation. The tumor was soft.

A PRINCIPLE, HITHERTO UNDESCRIBED, OF THE PHYSIOLOGY OF MOVEMENT AND POSTURE. THE PRIMITIVE SPINAL INTEGRATION OF MOVEMENT IN VERTEBRATES. DR. WALTER M. KRAUS.

At the present time we describe movements in such terms as extension, abduction, internal rotation, etc. In other words, we are describing activity of a neuromuscular mechanism in terms of the muscular part of that mechanism alone, leaving out the neurologic element of that movement; leaving out mention of its control by the spinal cord and its peripheral nerves. When we describe a complicated movement guided by integrations of the nervous system at levels higher than the spinal cord, we omit consideration of the final neuromuscular mechanism. It would seem wise to add some terms to those describing muscular movements which would give a notion of the embryology of these muscles as well as their innervation. This can be done very simply.

The nervous system is unquestionably built up of a series of physiologic levels, of which the higher or more recent ones, phylogenetically speaking, carry out functions which are different from the lower ones. It is my opinion that we must have some conception of the integrating action of the lowest levels in order to interpret the higher levels. We must know how the spinal cord integrates movements in order to understand how such higher integrations as the cerebellar act; and ultimately to understand why it has become necessary for these higher integrations to appear at all.

Properly to examine integration of movement in the spinal cord, it is necessary to consider in detail the general plan of peripheral nerves, the general plan of the innervation of muscles by them, and the embryologic grouping of muscles. I wish to present only the simplest facts in connection with these structures, introducing no theoretic considerations.

A typical thoracic spinal nerve having nothing to do with the limbs, shows a number of divisions after union of the posterior and anterior spinal roots. The trunk so formed divides into an anterior and posterior division. The anterior division divides further into a lateral and an anterior branch, the lateral branch dividing into a dorsal and a ventral.

As far as the axial musculature is concerned, the portions of it developed from the ventrolateral portions of the embryo are supplied by the anterior division and its branches, while the posteromusculature is supplied by the posterior branches. Thus a simple scheme results from a physiologic point of view, in the notion that general movements of extension are guided by the posterior division, while flexion and lateral movements are guided by the anterior division. When we apply this to the limb plexuses, we find ourselves confronted by two theories. Before stating them, it is necessary to emphasize that the limb plexuses do not include any part of the posterior division. One theory assumes that the plexuses are made up of the entire anterior division, the other that they are made up entirely of the lateral branch of this division. For the purposes of this discussion it does not seem

at the moment very important to try to reach a decision as to which of these theories is correct. I shall assume that the plexus is made up solely of the lateral branch, proceeding to discuss the details of my problem in these terms.

The primitive extremities of the embryo are flattened buds having a dorsal and ventral surface. The premuscle mass on the dorsal side gives rise to muscles the majority of which, in the adult, have an extensor function. The premuscle mass on the ventral side gives rise to muscles the majority of which have a ventral function. The crux of the whole situation lies in the fact that when considering the simplest function of a muscle, we find certain muscles have a function opposite to that which would be expected from a knowledge of the side of the limb bud (ventral or dorsal) from which they develop; for example, the iliopsoas group develops from the dorsal muscle mass of the lower limb bud but has a ventral function, that is flexion.

To return now to the question of innervation: We find that the embryology of the muscles is beautifully emphasized by the innervation of these muscles. It is well known that the nerves of the brachial and lumbosacral plexuses divide into two definite groups; one of which is posterior or dorsal and the other anterior or ventral. In the arm the ventral branches of the brachial plexus innervate the subclavius and pectoral muscles as well as all the muscles supplied by the musculocutaneous and ulnar nerve. What we find when we make a detailed table of muscles based on (1) whether they have been derived from the dorsal or ventral aspects of the limb bud, and (2) whether they are supplied by dorsal or ventral branches of the limb plexuses, is that those muscles which develop from the dorsal layers of the bud are supplied by the dorsal branches of the limb plexus. The analogy to the condition in the axial musculature is obvious.

From all this we arrive at some possibility of schematization. In the nerves and muscles of the upper limb we find, as in the case of the iliopsoas mentioned before, that a muscle which would be expected to have an extensor action has in reality a flexor action. In other words, were we to group the muscles acting in any given movement merely on the basis of their activity as muscles and leave out of consideration their embryology and activating nerves, we would be leaving out entirely the division of these nerves and of their end organs, the muscles, into a large ventral and a dorsal group.

To illustrate how this information may be used for discovering just how the spinal cord may integrate movement, let us take the process of stepping. The extreme forward movements of the leg are carried out by the iliopsoas, quadriceps and tibialis anticus muscles. Though this forward movement of the leg as a whole is spoken of as flexion, it is quite obvious that it is dorsal action since all the muscles which carry it out are of dorsal origin. On the other hand, the carrying backward of the leg is brought about by the hamstrings, gastrocnemius and soleus group and the tibialis posticus, all muscles having a ventral innervation and being of ventral embryologic origin. Consequently the movement of extension of the leg as a whole at the hip is, in reality, ventral in origin. To go further and take the associated movements of the arms and legs, when the left arm is forward and flexed, the left leg is extended at the hip, flexed at the knee and extended at the ankle. It is generally stated that the arm and leg on the same side are acting oppositely, in that the arm is flexed while the leg is extended, and the arm is, for example, forward while the leg is backward. If we interpret this position in terms of the neuromuscular mechanism controlling it, rather than in terms indicating the geometrical relations, such as flexion, extension, etc., we find

that the arm and leg on the same side are both directed, in this particular example, by ventrally innervated muscles, so that the notion that the arm and leg go in opposite directions on the same side must be reserved when the neuromuscular mechanism is considered as the basis of movement.

It is proposed that to the words now used to indicate the activity of a muscle such as flexion, abduction, extension, etc., one of two words be added, namely: ventrad or dorsad, which would serve to indicate three things:

1. The embryologic origin of the muscle.

2. The nerve supply of the muscle (ventral or dorsal branches).

3. The primitive direction in which the limb was carried by that muscle.

By adding these terms we describe movement in terms of the neuromuscular mechanism.

Furthermore, the primitive integration of movements of the spinal cord is seen to be one which throws into activity the muscles which develop together and are innervated together. This simple movement of a group of such muscles must be brought about by a correlation mechanism within the spinal cord, and forms the foundation of movement. So simple a movement as could be brought about by the simple activation of large groups of what were primitively ventral or dorsal muscles, was obviously insufficient for the needs of animals. Further and different integrations of movement became necessary and so not only the spinal cord but the more recently developed portions of the nervous system came into existence in order to make possible these new and more complicated patterns of associated movement. An example of one of these within the spinal cord itself is the flexion reflex, "flexion of the three great joints." This term gives the impression of a certain homogeneousness of action which, when interpreted in terms of the neuromuscular mechanism, is found to be entirely absent, in that flexion of the hip is a dorsad movement carried out by the iliopsoas group, flexion of the knee is a ventrad movement carried out, in this instance, by the hamstrings, and flexion of the ankle is a dorsad movement carried out by the tibialis anticus.

DISCUSSION

Dr. S. E. Jelliffe: I feel quite certain that Dr. Kraus has emphasized a method, or a new method, of analysis of motion, and whereas for myself the embryologic argument always lags behind the phylogenetic, I should like very much to have him link up the phylogeny of these movements and show wherein they conform to the present embryologic schemes. Then I think a certain number of the difficulties that he himself has suggested will perhaps be clarified. His presentation is most stimulating.

Dr. Kraus: I think the phylogeny of the matter is the only ultimate way to decide it. The reason for bringing these little animals down was to emphasize that point. I do not think, however, that we need to give the "why" of these things to emphasize the facts. The facts remain and I am dealing with facts, not theories.

Dr. Stookey's emphasis of the many functions of a single muscle is extremely pertinent. (Dr. Stookey's discussion was withdrawn.) We must take a primary function first and then try to explain if we can (and in many instances we can) why a muscle assumes a secondary function. Take, for example, the activity of the flexors of the wrist. There the prime action is flexion; but they have also assumed an action of adduction or abduction. We must examine these movements carefully before reaching conclusions.

Now, as to abduction, let us take the abductor of the thumb—that certainly is abduction; it is from the middle line, the axis of the extremity. But as I am speaking of terminology, I should like to emphasize that when we speak of it as abduction, we think of it in the same way as we think of abduction of the arm, though the innervation and embryology indicate that one is dorsad while the other is ventrad. If we use the terms ventrad and dorsad we know how different is the innervation in these two cases. I do insist (and I will make this the only point I would like to insist entirely on) that we must use the neuromuscular mechanism as a basis for the interpretation of movement and not the muscular only. If we are going to interpret the activity of higher levels, we must interpret that through the nervous system. We must not pass from the action of the cortex to flexion, for example, without knowing in the least how the spinal cord has come into that integrating action.

SOME THERAPEUTIC EXPERIENCES OF MYASTHENIA GRAVIS.

DR. CHARLES L. DANA.

The prognosis of this disease as generally given is very bad; according to Oppenheim, twenty-six of thirty-eight patients died; Dr. Hun found that most patients die in from one to three years; Dr. M. Allen Starr found that about 45 per cent. of the cases collected by him died within six months. My experience had been quite different.

I have had under observation fourteen cases during the last twenty years. I have not been able to follow up all of them, and some have died from natural causes but none of them has died of the disease while under my observation, and I have watched them for from one to seventeen years.

DURATION OF DISEASE WHILE UNDER OBSERVATION

1 year	5	7 years	1
2 years	1	12 years	1
4 years	1	13 years	1
5 years	1	17 years	1
6 years	1		

RESULTS

Recovered	4	Died of cancer	1
Practically well	3	Not followed up	1
Improved	4		—
Not improved	1		14

I think that the better prognosis of the cases was due, in part at least, to the treatment. This consisted of massive doses of strychnin combined with rest and elimination. The doses reached in some cases ⅓ grain given hypodermically two or three times a day.

In 5 per cent. of my cases the serious attacks were preceded by short attacks lasting only a few weeks and characterized by some ocular symptoms and bulbar weakness. In some cases, these prodromal attacks were not followed by any other attack for many years; in one case as long as seventeen years elapsed. I think it quite probable that mild abortive attacks characterized by diplopia and slight exhaustion of the bulbar centers are not infrequent. In some cases, the prodromal seizure was simply an ocular one, and one of the causes of casual ocular palsies of permanent type undoubtedly is myasthenia gravis. This is not recognized in the systematic works on ophthalmology. Myotonia is an occasional complicating symptom of myasthenia. I think the disease is toxic and endogenous and one that affects both

motor neuron and muscle. I have seen no result of note from endocrine therapy.

DISCUSSION

Dr. Bernard Sachs: I am willing to endorse Dr. Dana's rational therapy. In the cases we have had at the hospital during the past ten years, the uniform treatment, particularly for the bulbar symptoms, has been the use of strychnin, but we have not employed it in any such doses as Dr. Dana now recommends. I shall be very glad to try the high dosage.

Dr. Dana's experience has been extremely fortunate. Of all the cases in which I have made the diagnosis of myasthenia gravis pseudoparalytica, only a few patients recovered or remained alive long enough for me to record a temporary recovery.

I wonder whether Dr. Dana has not had the same experience that I have had in the course of these years: first, that the disease that some of us style myasthenia gravis includes a number of rather discordant types of disease, particularly in view of our experience during the past few years with various forms of encephalitis, not only epidemic encephalitis. There are no doubt other forms of infectious encephalitis besides epidemic encephalitis, and I am inclined to think that some of these cases covered by Dr. Dana may have been cases of that type and not cases of myasthenia gravis. The entire subject still remains more or less of a mystery to me, except that I have no doubt that there are cases that are due to severe toxic products circulating in the body. It would be only a short step from that to the supposition that some of the cases were due to infections of all sorts in the body, and I believe that further study probably will prove that many of these cases are really distinct forms of encephalitis.

If I were to state a general impression as to the number of cases I would be willing to call myasthenia gravis, I should say that I had not seen more than six or ten of them in so many years.

Will Dr. Dana state whether the symptoms cleared up rather promptly on the large doses of strychnin?

Dr. L. P. Clark: A number of years ago I was particularly impressed with the enormous advantage that Gowers gained from the use of strychnin in myasthenia gravis. Later, I noted cases in the service of Dr. Starr but recall no advantage obtained with either small or large doses. I continued to use strychnin for two or three years, beginning with small doses and running up to ¼ grain. How does Dr. Dana explain the action of the strychnin, if this is some form of glandular disturbance? How does the strychnin operate against an organic conductivity of the systemic values?

I have been very much surprised to note the enormous suggestibility that these patients have. Several months ago I was asked to see a patient because there was possibly a neurosis superadded. The diagnosis seemed quite correct and everything had been done for the patient that was possible. I suggested a change of environment by taking a rest in the country, and was much surprised the other day when this man's wife reported that he was walking two miles, was engaging in all sorts of activities, and everything but his speech (which was predominantly bulbar in type) was enormously improved. I was particularly impressed that this patient was going through a rather definite, almost psychotic delirium and I was surprised to see the type of mental reactions he was taking.

Dr. Smith Ely Jelliffe: While listening to Dr. Dana's paper, my thoughts have run along the lines that Dr. Sachs has so well emphasized: That whereas one can predicate a pure type of reaction focused about what is

termed "myasthenia gravis," shading off from such a picture a great number of semi, demi types are observable, some of which undoubtedly belong with the groups which Dr. Sachs has emphasized. There are also others that heretofore have been called periodical palsies, and a whole group of phenomena related to irregular muscular reactions due to acute or chronic conditions chiefly located in the medullary nuclei. It seems to me that that is a useful way of looking at the whole situation. Of course, if, with a certain trend of mind, one is apt to lump all these things together and speak of a disease *sui generis*, then I think Dr. Dana's attitude of mind quite justified. However, if one is inclined to see so-called pure types and divergent types and a whole group of situations which clinically may be grouped about a definite syndrome, it seems to me the results which have been reported are quite comprehensible.

Dr. Dana has spoken of the endocrine situation. It seems to me that Dr. Dana sketched too broad and glaring a type of generalization. I feel disposed to think that if the particular group of glandular involvements had a certain specific action, either on one or the other branch of the vegetative nervous system, then one could commence to formulate endocrine disorder. That is, one might say, "Here is a disturbance of one or another nervous arc in a muscular mechanism which is doubly innervated." The sympathetic arc of the vegetative mechanism apparently is the one that is most primarily involved in the *pure* types of myasthenia gravis. If that be so, there is a good rationale why strychnin should be of value in that type, because it is, of all of the plant alkaloids, the most powerful stimulant to the sympathetic branch of the vegetative nervous system. That is also a reason why calcium is therapeutically of value, and why sometimes this type of myasthenia gravis cases appears closely correlated with parathyroid disturbance.

Dr. B. Rosenbluth: In studying the literature of myasthenia gravis I came across a similar disease occurring in Switzerland and also in Japan, which offered all the symptomatology of myasthenia gravis. This disease occurs in the most crowded quarters and among the poorest class of people who are undernourished. In Japan it is found among the laborers in the rice fields.

I have had only four cases of myasthenia gravis, and instead of using medication, I have put the patient to bed and fed him by means of the duodenal tube. Dr. Larkin took cultures from one of my cases and one other but was unable to discover anything definite. Two of my patients recovered. We can see that any form of treatment that is kept up long enough, with stimulation and nourishment, will bring the patient over the bad part of it for two or three years.

Dr. Dana: Does this disease keep on recurring every year?

Dr. Rosenbluth: It recurs every two, three or four years.

Dr. Charles E. Atwood: The disease renews itself frequently by fatigue. Possibly the recurrences are from the suboxidation which takes place in fatigue. I have a case now that has lasted since 1907. The patient has done best on hygienic symptomatic treatment and small doses of strychnia. The myasthenic reaction is one of the most reliable tests, perhaps, in differentiating myasthenia gravis from other diseases.

Dr. Foster Kennedy: My experience has been that there is rather a difference of prognosis in the subjects who are young; that is, in the subjects who are under and those over 40, it being decidedly worse in those who are in their twenties. The disease, as I have seen it, has run a very much shorter course in the young than in the middle-aged or aged.

I have seen a series of necropsies on myasthenia gravis, with the extraordinary chance that in five of them an enormously enlarged thymus existed. This thymus was not so great as regards glandular bulk, but it was great as regards glandular expansion. The thymus was perhaps two-thirds the size of my hand, stretching down over the pericardium—an extraordinary phenomenon, one which certainly cannot be without significance in the disease, though its exact correlation with the symptoms I am not able to state.

Dr. Charles L. Dana: With regard to the point that Dr. Sachs made, I had written this—I did not read it: "Myasthenia gravis is a disease which runs peculiarly true to type," etc. Therefore, I think that Dr. Sachs' suggestion that we ought to scrutinize these cases with especial care is a wise one. One case I had in mind was perhaps not true myasthenia gravis, though I thought it was at the time. We do not have to give massive doses of strychnin always or all the time. I find sometimes that 1/15 grain is enough, and sometimes even a little less; but it seems to me that the most permanent and satisfactory results were those in which these larger doses were given for a considerable time.

The symptoms do not clear up immediately. You will have to use the injections for perhaps three or four weeks before they begin to show results. It seems to me that there is nothing physic in the patients. The patients were treated seriously, of course; they were put to bed a large part of the time and they were kept very quiet; they were given these large doses and they knew they were being actually treated. There was not any suggestion about it, nor were there any psychic elements in the case.

I do not quite understand Dr. Jelliffe's remarks. It seems to me the disease is a poison of the somatic rather than of the sympathetic part of the system.

Dr. Jelliffe: But the somatic muscles have a double innervation.

Dr. Dana: But it is the somatic part of the innervation that is affected.

Mr. President, in regard to the point that you made, I can only say that one of my patients, one of the very best ones, a boy of 19, has now been well for six years; but all the other patients were in the thirties or forties—perhaps more of them beyond 40 than under it.

The Three Hundred and Ninety-Second Regular Meeting, Dec. 6, 1921

Dr. Foster Kennedy, *in the Chair*

SYNDROME OF THE RETROPAROTID SPACE, WITH SPECIAL REFERENCE TO A NEW INTERPRETATION OF SOME FORMS OF FACIAL PARALYSIS. Dr. Byron Stookey.

Dr. Stookey presented a patient showing a pure syndrome of the retroparotid space, as described by Villaret (1916). Though the injury had been suffered sixteen years previously, complete paralysis of the glossopharyngeal, vagus, spinal accessory, hypoglossal and cervical sympathetic nerves still remained, with ptosis of the eyelid on the same side, miosis, narrowing of the palpebral fissure, enophthalmos, loss of taste on the dorsal third of the tongue, difficulty in swallowing and eating, paralysis and atrophy of the tongue, and weakness in elevation of the arm due to paralysis of the trapezius. Attention was called to the development of the clavicular portion of the

pectoralis major and the clavicular head of the deltoid, both of which stood out prominently on elevation of the arm beyond 115 degrees. These two muscles thus compensated for the loss of rotation of the scapula in elevation of the arm.

Oblique withdrawal of the dorsal wall of the pharynx toward the sound side—"mouvement de rideau" of Vernet—was demonstrated; but Dr. Stookey did not believe that in this patient this sign could be taken as a sign of glossopharyngeal paralysis in view of the other nerves involved. Vernet's view that the vagus is a purely sensory nerve was denied, in view of the histologic character of the dorsal motor nucleus of the vagus and the nucleus ambiguus as shown by finer histologic studies of the nuclei of the vagus following section of the vagus. Its efferent character, as well as afferent, was emphasized. In Dr. Stookey's patient a slight facial weakness when the face was at rest was noted, though the facial nerve was intact and there was no weakness in voluntary movements. There seemed to be rather a loss of tone. In view of the fact that striate musculature possesses a dual efferent innervation, this loss of tone, it was believed, might be due to loss of the efferent sympathetic fibers to the facial musculature, because of injury to the superior cervical sympathetic ganglion.

DISCUSSION

Dr. Smith Ely Jelliffe said that he was delighted to hear Dr. Stookey raise the point of the sympathetic innervation of the musculature in the myasthenic reaction. He believed that Dr. Stookey had been a little overcareful in advancing his hypothesis of double innervation, and that he had stuck too closely to the anatomic evidence alone. It seemed that the splanchnic remnants were over-represented in the facial group.

Dr. I. Abramson believed that the dual innervation might account for the frequency of contractures after facial palsy. This occurs more often than with any other cranial nerves. While explanations of contractures have been many and varied, none of them seems fully to account for the phenomena. A second corollary is the occurrence of the facial hypotonia, seen in tabes, causing sagging of the features. It is known that the cervical sympathetic nerve is often attacked in tabes. Facial contracture can be regarded as a tonic spasm on account of the loss of normal equilibrium between the clonic and the tonic parts of the musculature.

Dr. Jelliffe added that he referred to Winkler's explanation of the sphenopalatine as a new nerve, and that that probably had considerable relevance to the discussion of this region. He noticed in the case presented that the tongue was very red and rigid. The salivary innervation was also involved.

Dr. Foster Kennedy said he was surprised that Dr. Stookey laid such emphasis on the comparatively recent recognition of the syndrome as an entity. It surely preceded the date that Dr. Stookey gave. He asked whether this condition was not diagnosed by many of the members as early as 1915. He said he recalled a case in the Neurological Institute, in 1910, in which syphilitic involvement of the skull gave evidence of a lesion clearly recognizable as that described by Dr. Stookey. In the war, too, cases were noted with frequency. The sign of glossopharyngeal paralysis by deviation of the uvula had been recognized since 1907. He referred Dr. Stookey to a paper by Maloney and Kennedy, in *Brain*, 1912, on the pressure sense in the face.

Dr. Byron Stookey (closing) said he could add nothing to the evidence in regard to the sphenopalatine. It was supposed that the supply of the parotid through the ninth nerve was from the *nucleus salivarius* to the otic ganglion

and thence by the auriculotemporal nerve. The preganglionic fibers were from the ninth nerve, and the postganglionic fibers from the otic ganglion through the auriculotemporal to the parotid.

In answer to Dr. Kennedy, he said that there was no reference earlier than Villaret, in 1916, to the retroparotid space as a locus of extracranial injury of those nerves. The syndrome of Hughlings-Jackson is not extracranial. Tapia and Schmidt refer to lesions involving the nucleus ambiguus, but these are intracranial, not extracranial injuries. He asked whether Dr. Kennedy's case involved the cervical sympathetic nerve. Dr. Kennedy had referred to deviation of the uvula, but this was, of course, totally different from deviation of the posterior wall of the pharynx due to paralysis of the superior constrictor muscle. In paralysis of this muscle, the pharynx is drawn obliquely over to the sound side—*"mouvement de rideau."* He asked whether Dr. Kennedy's own work did not deal exclusively with the afferent innervation of the face, and not with the efferent. If Dr. Kennedy's work referred to a dual innervation, it antedated all other work in this line by quite a few years.

DR. KENNEDY said that his work did not conclusively show this point.

DR. STOOKEY said he was pleased to know that Dr. Kennedy's work dealt solely wth the afferent side, as he wished to emphasize that his paper that evening referred to a dual efferent innervation, not afferent.

A PSYCHIATRIC STUDY OF SUICIDE. DR. THOMAS W. SALMON.

Dr. Salmon presented the human, rather than the professional, side of the problem. It has been stated that there are few people who at some time or other in their lives have not thought of self-destruction; but it is not easy to see exactly what is meant by that statement. It is true that most people, in childhood, have had visions of being laid out in the best chamber, surrounded by weeping relatives and members of the family who are overcome with grief at their former lack of appreciation of the departed. Thoughts of suidside are almost equally wide spread, and most people who have left autobiographies have spoken very definitely of suicide; but these thoughts are usually disposed of by the mechanism, which we possess, to dispose of ideas which interfere with our happiness. The relative infrequency of suicide accomplished, as compared with thoughts of self-destruction, bears witness to our power of preventing this type of casualty.

Apart from the human standpoint, suicide is of interest as a psychiatric study: first, because it is due to a preventable phase of mental disorder; and, second, because it involves questions of therapeutics and of hospital management. Recently, suicide has taken on a new and wider interest, and it is from this larger standpoint of methods rather than material that the present study was undertaken. Not only were cases considered which were allied to frank mental disorder, but also those in which this strange disturbance of human conduct occurred in sane persons.

Dr. Salmon took as an associate in this study a well-trained social worker, and tried to follow up every case. A period of six months was taken for intensive study which presented many difficulties, because it was not always possible to precede the undertaker or to follow the police, or to make oneself welcome in every home; but every available channel of evidence was used.

The successful collection of minute details was due to splendid cooperation on the part of medical examiners. It was found that the gas companies were those next interested, whether on account of the amount of gas wasted, or not, is not clear.

Generally speaking, it would seem to be a simple matter to determine whether or not suicide took place; but those who have studied the subject tell us that probably not more than two thirds of the cases of suicide are reported, and that many cases are mistakenly termed suicide; also many cases are associated with homicide, so that a double toll of lives has to be estimated. Any personal stress or economic stress may be associated with suicide; but no one knows the exact proportion of these cases.

Sentiment which interferes with these investigations may be of two kinds: In some cases, the relatives want the suicide made widely known; this usually has to do with various religious procedures which are modified in case of suicide. On the other hand, every effort is made to assign the death to natural causes instead of to suicide. For this reason, one sometimes gets better evidence from neighbors than from relatives. Dr. Stearns found a typical illustration of this in one case of a very old man who committed suicide. The relatives were reticent about giving information; but a carpenter, who lived next door, told the physician that he knew the old man would commit suicide because he sat for hours in the carpenter's yard without speaking and when he spoke it was to complain that his bowels ended in the middle and that a man in that condition ought to be dead. The family physician confirmed this evidence.

Suicide from suggestion is not uncommon. One very old man traveled all the way from Chicago to hang himself in a certain barn, because it was the custom for members of his family to do so, and he wanted to commit suicide in the orthodox way. The obvious cure in a case of this kind would be to burn down the barn and prevent further incidents. In New Orleans, there is a "suicide oak" which no doubt has helped, with the aid of suggestibility, to put many persons out of the world.

The statistical study of suicide shows that it is steadily on the increase in all American cities. Many theories have been advanced, but the one most tenable is the steady drift of the population to the cities. Why suicide should be more common in cities needs careful analysis. Perhaps availability of weapons has something to do with it, as has also the ready supply of gas. Dr. Stearns found a very striking relation between depression and suicide; he found also that the suicide rate rises with every decade of life in a uniform manner. In the eighth and ninth decades, it is remarkably high. He found a high rate among the Irish, in contrast to the low rate found by other workers. Among the foreign born the rate is higher. This reflects the greater economic stress in this section of the community.

Factors of medical interest are the relation of suicide and mental disease: In 42 per cent. of the cases there is a history of mental disorder, and if we include psychoneurotic cases this rises as high as 58 per cent. Seventy per cent. of all cases of suicide in which there is mental disorder occur in the manic-depressive groups; in 23 per cent. there are other psychoses, making a total of 93 per cent. of mental disorders in which depression is a marked factor. Contrary to the usual idea, there are often depressive cases and suicides among psychoneurotics. If it were not for the fact that most patients with mental disturbances are hospitalized and guarded, the number of suicides in this group would be alarming. In a group of this kind, 157 in number, which was studied, seven were among children. Child suicide has been made a study by itself; but Dr. Stearns secured full data in these cases, and it was surprising what abnormality existed long before suicide occurred.

This wealth of descriptive material points to definite measures of prevention. The suicide death rate is higher than that for typhoid fever. We spend large appropriations for the prevention of typhoid, but we do nothing to prevent suicide. The very first preventive measure of importance that occurs to one is the necessity of recognizing depressions in their earliest stages. This is a responsibility for the public and for the medical profession. A distinguished surgeon in Chicago told Dr. Salmon that a minister of his acquaintance was fond of coming into his office, and wasting the physician's time by telling him how worthless he felt himself to be, and what a discredit to his profession. The surgeon said to him, "If I felt like that, I would go to the lake and walk east till my hat floated." Finally, this is exactly what the minister did. The suggestion was the final link between depression and suicide. The danger is greatest in the early and the late stages of manic depression. In the severe stages the mind does not focus clearly on the suicidal idea.

In one case recalled, that of a magazine writer, the man came into Dr. Salmon's office and introduced the topic of what became of unclaimed bodies found in the East River. He said he had a professional interest in the problem. The man had a pallid, unhealthy appearance, and it was suspected that he was suffering from depression. Dr. Salmon told him that the bodies were distributed to the various dissecting rooms for the use of medical students. Feeling that confession might pave the way to relief, he went on to speak frankly of the subject of suicide in general and to sketch an outline of the questioner's probable mental symptoms. This produced a response on the questioner's part and he admitted that he was suffering from acute mental depression. He was told that his symptoms were extremely susceptible to treatment and was urged to go to a hospital to be taken care of. Arrangements were at once made for his reception at Bloomingdale and he entered the hospital that evening. He expressed himself as well satisfied, and stated that he felt very much safer when he saw the nurse patrolling the corridors at night and he was able to rest comfortably. Unfortunately, his brother came from Chicago and was very indignant that the patient had been placed in an institution by a "fool doctor." He removed his brother with the idea of taking him home. It was stated that on the journey home the patient appeared unusually light-hearted and facetious. The brother was positive that the patient was much better. On arrival, the patient made an excuse to go to the bathroom for a few minutes and when there cut his throat. This case illustrates the absolute necessity for care in the early depressive stages, and indicates clearly the need for early treatment.

During the war, statistics showed that war must have been in some measure a substitute for suicide. The A. E. F. figures were 5 per 100,000, in contrast to the regular army figures of 53 per 100,000. This large difference probably needs many causes to explain it. One factor may be the exclusion of those persons that are obviously suffering from mental disorders. Hospitals were available from the front line to the base for early mental symptoms, and the slightest abnormality was reported early. Probably the treatment was better in the Argonne than in New York City today.

We have very powerful resources for the prevention of suicide. Many patients who attempt suicide give positive evidence of a long period of conflict before the act—between the desire to die and the instinct to live. The Reverend Dr. Warren has instituted what he calls the "Save-a-Life League." He answers telephone calls from people about to commit suicide. He has

some one on hand, day and night, and by entering the field from a different angle, that of religious and moral counsel, he saves people at a critical time. He has a wonderful collection of letters and histories testifying to the results obtained. Probably the most powerful aid is the factor of human friendliness, which is so often lacking in our medical treatment of these patients. One is impressed by the extreme loneliness and isolation of the sufferers seen at Bellevue Hospital after being rescued from attempted suicide. A powerful human resource has not been put into use. In Japan there exists an anti-suicide league, and on the railway crossings there are signs suggesting that these should not be used for suicidal purposes. Whether the suggestion works in the direct or the reverse fashion is doubtful.

The suicidal intent often lasts for years and years before consummation. In Wisconsin, in an insane asylum, a man built an extraordinary barn, with fantastic cupolas. He kept adding one after another, and when he was unable to add another, he hanged himself on the last one. The constructive work had probably been a sort of safeguard, and when this was exhausted, he took his life. Had the architectural standards of Wisconsin allowed of an indefinite number of cupolas, he might have lived to old age. In another case the safeguard proved to be an altruistic reason. An insane man, while walking with a new attendant, tried to jump into the river. The attendant cried out anxiously, "Don't do that or I shall lose my job." This plea deterred the suicide at the moment of impulse.

In regard to other preventive measures, Dr. Stearns suggests that something might be done to lessen the extreme suggestibility of those intending to commit suicide. While the world cannot be adjusted to the psychopathic mind one can place some restrictions as to suggestion on the movies and the drama. Fire-arms and gas can be safeguarded. The fact that there is a diminution of suicide by drowning in winter, on account of the freezing water, shows that availability has something to do with it. The policy of avoiding frank discussion with the patient seems to be a poor one. Often confession proves helpful. The details of general statistics tell little, and the method for future study is the carefully worked out case method from histories of patients who have been rescued. The time for prevention is during the early and late stages of depression when there is a conflict between the desire for life and the choice of death.

DISCUSSION

Dr. Smith Ely Jelliffe said that the subject was so large that many points have, necessarily, to be left untouched. The metaphor of the barn, used by Dr. Salmon, could be taken as an illustration of a type of reconstruction process which takes place in the minds of many neurotic patients. The construction of psychologic barns can be seen as a protective mechanism. If this fact is not recognized, the patient may, in the end, get beyond protective mechanism. Another important point is the recognition of the fact that what may appear as a psychoneurosis on the surface may be the early stage of a severe mental disturbance. The psychiatrist must endeavor to change the mode of expression of the patient into something beneficial. Delusions represent ego-protective devices, and they may be piled up more and more, one above the other. At times these delusional formations of pronounced grade may be replaced by religion, mysticism, or other expression of a less *outré* type. The early and late stages in manic-depressive patients are particularly dangerous. In the early stages they are suicidal, not having built up their delusional defense mechanisms; in the later stages, the patient has come through a rich delu-

sional stage and has to be protected until he gets back to his normal. In regard to methods, the analytic method is often much condemned because so frequently misunderstood; but it may be helpful at a certain stage and more often saves the patient from the river than it drives him to it, as some superficial and nonpsychiatrically trained physicians often claim. To frighten patients away from a competent psychiatrist is as criminal as it is to allow nontechnically trained people to treat mental illnesses.

Dr. S. R. Leahy remarked that while dementia praecox is not usually thought to be associated with suicide, this is not an infrequent combination. When we consider the type of hallucinations it is surprising that more suicides do not occur. The patient may endeavor by suicide to escape the imagined dangers threatening him. The dementia praecox patients that commit suicide are impulsive in type. They do not speak of the intention and evade the issue. If once prevented, they become more determined to commit suicide. In persons with marked mental and motor retardation, suicide is unlikely. They cannot plan self-destruction. The fact that families do not want to put depressed patients in hospitals often leads to suicidal attempts. They are not properly guarded. Patients with alcoholic hallucinations are liable to commit suicide on impulse, to escape their terrors. They are likely to jump off ferry boats or perform similar acts. Any condition, organic or functional, producing depression, makes suicide likely. This is seen in arteriosclerotic or general paresis.

Major Jarvis (by invitation) said that the profuse advertising of every suicide case, with its appeal to suggestion, is to be deplored. Considering that environment in rural districts was a factor, Bismarck drew settlers from remote districts for his rural communities because of the large percentage of suicide. Environment is an important factor, as evidenced by the frequency of suicide at a post in Northern Arizona, called, in the army, "Suicide Post." Four officers attempted suicide there, and even one Indian, the latter being a very unusual occurrence. This post is 110 miles from a railroad and is situated near a dark canyon at the foot of a high lava mountain which shuts out the sunlight at 4 p. m. This gloomy environment is doubtless a contributory factor. Bluntness is sometimes helpful to dissuade from suicide. This is illustrated by the story of a rough and ready old army colonel in whose command a suicide epidemic appeared. He called for one man suspected of contemplated suicide and roughly told him that if he wanted to shoot himself he should go out on the lot and do it instead of messing up the barrack room floor. The result was that the man decided not to commit suicide.

Dr. L. Pierce Clark believed that the personal morale and sense of identity, in the country, constitute a factor against suicide, as compared with looseness of standards in the city. He had been impressed with a peculiar psychology in some patients with depressive conditions. They seem to have a sense of separation from God, by reason of sin, and plan suicide with expiational intent. If they are rescued, and if the intent was sincere, they feel that they have done, as it were, all they can, and have thereby atoned. Thereafter they get well. If they do not make a sincere attempt, they still feel they have to go through the ordeal, and they do not improve. Might this not suggest the advisability of letting these persons really attempt suicide, taking precautions to rescue them, so that they can gain this sense of rehabilitation which helps in their cure? This should be carefully studied. Many patients at Bellevue confess that no one is left who really loves them. This

means that the out-stream of the libido has lost its attachments and has resulted in attempted suicide, an extreme gesture of despair.

Dr. Kempf was inclined to take issue with Dr. Salmon regarding methods. He believed that often the part of the body is attacked which is associated with some pain or discomfort, as, for instance, in the case of a man who complained of intense headache and who shot himself through the head; and one young woman who suffered precordial pain who shot herself through the heart.

Dr. Wechsler said that in some cases, such as the hebephrenic types, the impulse has no logical basis.

Dr. Foster Kennedy said that in the war men who wanted to commit suicide often put themselves in murderous positions in the front line. In Europe the method by drowning seems more popular than other methods, and has no seasonal variation. From a study of the suicide rate in Saxony, compared with that of Ireland (358 against 17 per 100,000), it would seem that there must be something more than personal individual variation to account for such racial discrepancy.

Dr. Salmon (closing) said that the increase of suicide had no doubt something to do with the choice of methods. There is avoidance of mutilation by women and the preference for fire-arms by men. In the army shooting is often chosen because it is considered more or less a soldier's death and somewhat removes the stigma. In regard to the story of the blunt colonel, doubtless the frankness was useful in preventing the issue. In regard to the A. E. F., it must be remembered that only 60 per cent. of the men were actually under fire. Some general cause must have operated to lower the suicide rate, since after the armistice it resumed the former proportion. In regard to loneliness in the country, that cannot be compared to loneliness in a large city. There are at present 75,000 ex-service men in New York whose homes are distant. That must have some effect in increasing city loneliness. Nonmental cases are of peculiar interest. The subject is very large and it is impossible to touch more than the outline. The best method of study is the careful detailed case study of the patient who has been rescued from attempted suicide. There rarely is a second attempt. Something has occurred in the life of the patient along the line suggested by Dr. Clark. Often the person who has attempted suicide finds his first friends in the hospital. He is clapped on the back and the friendly democratic atmosphere increases the homely human resources of the hospital and adds to its therapeutics. The days of statistical study alone and of the measurements of skulls and ears are past. When we carry out careful detailed study of living cases as a routine measure we shall know more about the psychology of suicide.

IODIDS, OBSERVATIONS ON THEIR USE AND VALUE, INTRAVENOUSLY. Dr. Frederick J. Farnell.

Dr. Farnell asked whether tissue and cell changes could be more rapidly brought about by giving a highly concentrated solution of sodium iodid directly into the blood stream; whether the same result could be obtained by giving a concentrated solution along with a bland syrup, by mouth, and whether the apparently good results depend on the high concentration or on the intravenous channel of administration.

Iodin in the form of an iodid can be given in hypertonic form intravenously. When given into the blood stream in hypertonic form iodin has a tendency to reduce the idiosyncrasy toward iodism. Iodids introduced into the blood

stream appear to readjust systemic fungus disturbances (oidiomycosis) very rapidly. Iodids given intravenously in concentrated form appear to help materially the action of arsphenamin on the diseased tissue and cell. Iodids introduced into the blood stream in hypertonic solutions probably have some influence in reducing the edema, hyperemia, etc., of the brain in increased brain bulk disorders.

DISCUSSION

Dr. Smith Ely Jelliffe remarked that in recent years he had noted definite restoration of the subject of pharmacology and therapeutics to a place of respect in teaching and a renewal of interest in the action of drugs on the human body. Pessimism is receding and a more healthy pharmacology advancing. The rôle played by iodin in the body is extremely important. The iodin mechanisms are extremely intricate and only partly analyzed, the chief question being the iodin content of the thyroid. He said he had hoped that Dr. Farnell would touch on the action of iodin on the sympathetic arc of the vegetative nervous system. The vegetative nervous system is definitely affected by iodin, chiefly on the sympathetic side. This, in turn, influences body metabolism. Dr. Farnell had approached the subject from a different angle—that of specific disorders.

Dr. J. H. Leiner said that in regard to the combination of iodin with arsphenamin medication, there is now a routine practice of this method in Vienna. It is seen that iodin enhances the effect of arsphenamin given intravenously.

Major Jarvis (by invitation) said that Dr. Nicholas Schenk of Chicago had noted the absence of tuberculosis and of skin diseases among the Eskimos. This is due to the large amount of iodin in the blubber and fish which form their staple food. These people fall prey to tuberculosis and respiratory diseases when they come to a temperate climate.

Dr. Foster Kennedy said that Mr. Hoover put an end to the epidemic of adolescent tuberculosis in Belgium by increasing the fat ration.

Dr. Farnell (closing) said that this work was experimental. He first tried giving large doses of potassium iodid intravenously; but it caused thrombosis from irritation of the vein lining. He then tried injection of 400 grains (25.7 gm.) of sodium iodid in a case of oidiomycosis. The patient was comatose for a day, but recovered without any ill effects. The man was cured of lesions of the lung and of the central nervous system. The sodium salt injection was apparently harmless.

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

Regular Meeting, Nov. 17, 1921

James B. Ayer, M.D., *President, in the Chair*

SCHIZOPHRENIC DETERIORATION: BIOCHEMICAL STUDIES AND PRESENTATION OF CASE. Dr. Karl M. Bowman.

The case I desire to present is that of a boy, aged 15 years, whose father had had a somewhat similar condition when he was this boy's age; but he has apparently recovered and has gotten along fairly well in life. This boy has never been rugged and has been afraid of other boys because they were stronger physically. In December, 1920, although no sudden change was

noted, the boy commenced to lose weight. He left school because of his weakness and loss of weight and later on came into the hospital. He has been rather quiet, self-centered and seclusive since an early age, and has been much wrapped up in his own thoughts, though it is not entirely clear just what these ideas are. The tendency to day dreaming apparently is not far advanced. He complains of dizziness, poor appetite, headache and various bodily symptoms. He has had fits of irritability, during which he has broken things, and also spells of crying.

Examination showed that he was not well developed physically and had poor chest expansion; his blood pressure was: systolic, 115, diastolic, 78. His fingers were long and graceful. He had no axillary hair and the pubic hair was of the feminine type. Results of examination by internists and by nose and throat specialists have been entirely negative. Laboratory tests showed that his basal metabolism on four different occasions was —24, —31, —37 and, following the feeding of thyroid extract, one grain a day for two weeks, —25. A roentgenogram of the sella was entirely normal. The blood chemistry was within normal limits. Spinal fluid, urine and blood examinations were negative. This case is taken as an example of investigating from the physical standpoint in order to correlate, if possible, certain physical findings with some of the mental states.

A further investigation along these lines was made on nine patients with dementia praecox, seven of whom had dementia praecox of the paranoid type and two of whom were hebephrenic. They all had cases of long standing. It was felt that laboratory studies over a certain series of cases might reveal to us the possibility of certain definite laboratory findings which would be constant in all cases. If not, we felt that consistently negative findings would be an equally valuable observation or even a marked scattering of results would be valuable. What we did was first to take twenty-four hour specimens of urine which were examined quantitatively for acidity, total nitrogen, ammonium nitrogen, uric acid and chlorids. Some of our findings were low and some were high, and there was no constant feature in the urine findings. This merely confirms Dr. Folin's findings as far back as 1904-1905. Next the blood chemistry was studied. We made quantitative examinations of the nonprotein nitrogen, dextrose, uric acid, chlorids and carbon dioxid combining power. In the nonprotein nitrogen test all but one fell within normal limits. In the dextrose test three were above normal limits and there seemed, perhaps, a tendency for the sugar to run higher than normal. In the uric acid test the findings were within normal limits. In the test for chlorids the findings, with the exception of two, were within normal limits, and those were close to it. The carbon dioxid combining power of the blood was within normal limits. The blood sugar tolerance curves showed great variation with no constant type of curve, but a considerable number were definitely abnormal. The basal metabolisms were ascertained. For two these were between 0 and + 3, and the rest fell below 0. One was —8. Three were between —10 and —19, two were —20, and one was —27. This we thought was of some significance, and in view of the case presented this evening it would seem that this point is worthy of further investigation. The renal function test was performed and showed the lowest result as 62 per cent.; all were, therefore, quite normal. The Goetsch test was negative in every case. The hemoglobin was tested in all cases, and the findings were entirely within normal limits. The red cell counts were high, the white normal. We felt that in reviewing these findings we had a definite tendency of the basal metabolism to fall below the

normal, and we had a large number of abnormal sugar tolerance curves but no constant picture. The other findings all seemed essentially negative.

DISCUSSION

DR. C. MACFIE CAMPBELL: Dr. Bowman's observations are of great importance because in these deteriorating cases we are dealing with a type of disorder which is of great severity, extremely frequent and of great social importance. After all, the greater number of patients for whom the state is responsible are those with cases of deterioration of which this boy's case is one type, and therefore it is one of our most important problems. The tendency in studying these cases has varied from time to time. If autointoxication was in vogue, the physician looked for autointoxication. Focal infections attained importance, and he took out teeth and pruned the individual in general. The *libido* later claimed his attention, and he traced the libido from its genesis to its maturity. There are few patients who have been studied in their entirety. As a rule, the presentation of a case has been from one aspect. Perhaps of the greatest importance at the present moment is the study of such patients in toto, to see if at the root of their emotional difficulties there may not be some disorder which we may formulate in simpler terms. Each man is different from his neighbors. Are we going to formulate his constitutional traits and maladaptations in complex terms of the emotional life, or can we find the key to these traits and to the disorder in metabolism or the chemical energies of the system? We study this patient from this standpoint and find that his basal metabolism is low. Is that one of the fundamental component factors which produce the total fault? In his emotional life is his limitation (lack of plasticity of the libido) largely due to the fact that some of the elementary components are lacking? The time may come when we shall not seek the etiology of these disorders in one popular formula or another but shall try to understand the constitutional endowment of the person both in its simple and complex factors.

DR. J. B. AYER: Has Dr. Bowman succeeded in raising the metabolism of these patients showing a low rate?

DR. DONALD GREGG: Has an early tuberculous infection been looked for in this case? Tuberculosis would give more or less the same picture.

DR. BOWMAN, in closing: In only one case did we try to raise the basal metabolism. We started with 1 grain of thyroid, and then the patient decided that yeast would do him more good than thyroid, so we got no further. Regarding the interpretation of the sugar tolerance, I don't believe it can be interpreted. We have a number of different curves which are being variously interpreted. We know that in diabetes the sugar curve mounts up at the end of the first hour and does not return to normal at the end of the second hour. In hyperthyroidism and hyperpituitarism there is this diabetic curve. In hypothyroid and hypopituitary conditions there is the exact opposite type of curve, for at the end of the first hour there is almost no rise in the blood sugar, and at the end of the second hour it is entirely back to normal or below normal. I cannot see how one can make any interpretation of this great variation in findings. It is claimed by certain men that there is a typical curve in dementia praecox. This report does not confirm their findings. As regards the findings in early tuberculosis, my understanding is that there should be a certain amount of fever. In that case the basal metabolism would be definitely increased and not decreased. I have no knowledge of any work done in tuberculosis as regards sugar tolerance curves and assimilation.

HYSTERIA AND PERSONALITY. Dr. Martin W. Peck.

The patient presented showed personality disorder with recurrent hysterical states. The patient, a girl of 19, was admitted to the Psychopathic Hospital six months ago with nearly complete hysterical paralysis, aphonia, and a mental state of anxious, fretful hypochondria. There was a history of two similar attacks during a period of two years. After two months without change, she suddenly recovered and has since been in robust physical health and without somatic complaints. At the same time she developed a type of disagreeable behavior which has kept things in a turmoil, and has made it impossible for her to return home or to get on in other than hospital environment. She appears to derive her main satisfaction in teasing, ridiculing and interfering with those about her, but all is done in a mischievous and semi-playful, rather than malicious, manner. She also indulges in much troublesome horse-play, and is tiresomely talkative. Her past history proves that this conduct represents in a general way, her normal state since childhood. In play she did not get on with her mates; in school her unfortunate disposition was notorious; at home she antagonized the neighbors; and her working career included frequent changes of position and continuous petty friction of her own making. She showed lack of normal affection and absence of serious purpose in life. In appearance she was attractive and graceful; she had normal intelligence and considerable artistic ability. At no time had she been delinquent in the usual sense of the term, or shown tantrums of any order. While friendly enough toward physicians, it had never been possible to establish a sufficiently frank relationship to unearth the deeper factors in her character difficulties. The case was discussed from the standpoint of descriptive analysis and her personality defects outlined by grouping them in the fundamental spheres of reproductive, herd and self-preservative activities.

DISCUSSION

Dr. H. C. Solomon: I should like to ask Dr. Dewey and Dr. Walton what their attitude would be toward the commitment of this girl. Would they be willing to go before a court and ask for her commitment?

Dr. G. L. Walton: I should not be willing to commit her without further investigation. She appears to be a high grade imbecile. She has a hypermanic tendency, a lack of moral sense and pleasure in disturbing other people.

PRESENTATION OF CASES OF ORGANIC PSYCHOSIS (EPIDEMIC ENCEPHALITIS). Dr. Lloyd T. Thompson.

In the last two months we have seen at this hospital at least five cases in which the diagnosis of epidemic encephalitis is the most probable one, but owing to the fact that there is no epidemic of this disease at the present time and that these cases are somewhat atypical, the final diagnosis of that disease has not been made. The two patients presented are especially interesting because of the psychotic symptoms shown in addition to marked and changeable neurologic disturbances.

Number one is a man, 49 years of age, who was admitted with a complaint of tremor of the right arm and weakness of the entire right side. The onset was sudden, five weeks before admission, with dizzy spells, tremors, headache and dull pain over the entire right side. At one time there was blurring of vision.

He had a mask-like facies with weakness and hypesthesia of the left side of the face. The pupils were unequal and irregular but reacted normally. There was coarse tremor of the right arm and hand with weakness and incoordination on the right side. The deep reflexes were more active on the left side with a Babinski sign on the left. Reaction to pin prick was lost on the right side. The neurologic picture was very changeable from day to day. A double ptosis and paralysis of the left internal rectus developed. Repeated lumbar and cistern puncture showed only a consistent increase in the sugar content.

The patient was somnolent in the morning but became active toward evening. He became hallucinated and developed ideas of persecution. At one time he presented a typical manic condition with flight of ideas, elation, overactivity, etc. He was correctly oriented but showed impairment of memory and judgment.

Patient number two presented a similar history and changeable neurologic disturbances, but the mental condition was somewhat different. On admission his condition resembled very closely that in a case of catatonia with sudden outbursts of aimless excitement. A few days later his condition changed and seemed more like a depression. He admitted that he was very unhappy and worried, and there was marked retardation. The patient was hallucinated and had self accusatory ideas, but these symptoms have now disappeared.

These cases are reported to point out the possibility of sporadic encephalitis or the possibility that they may be the beginning of another epidemic. They also serve to illustrate the prominence of mental symptoms and the close resemblance to other forms of psychoses.

DISCUSSION

Dr. H. I. Gosline: Dr. Thompson said that the colloidal gold test was negative. What does this mean? It has been reported that the test did show some sort of a curve in cases of epidemic encephalitis.

Dr. D. J. MacPherson: To what extent is it true that increased sugar is present in epidemic encephalitis?

Dr. Harold E. Foster: In the series which we had at the Massachusetts General Hospital increased sugar was present in all our cases. In our fatal cases the sugar ran markedly high. It also ran very high in a case of encephalitis with acute retention. Syphilitic cases, especially general paresis, run fairly high but not as high as encephalitis, although they may overlap. In laboratory work it cannot be said that any one thing will make the diagnosis. I think the diagnosis of encephalitis can be questioned in any case that has run for any length of time with a normal spinal fluid sugar.

Dr. Thompson: The colloidal gold readings were actually negative except for a few changes to "ones." There is no characteristic reading for encephalitis. It may be anything from a negative to a "paretic curve." However, the changes that do occur are in the first five or six tubes and seldom in the tubes of greater dilution.

HISTOLOGIC EVIDENCE OF THE RESULT OF TREATMENT OF GENERAL PARALYSIS. Drs. Harry C. Solomon and Annie E. Taft.

The object of this investigation was to determine whether treatment of cases of general paralysis produced any effects that might be observed histologically. For this purpose a histologic examination was made of the brains from twenty-seven patients who had had general paresis and to whom treat-

ment had been given. These were compared with the brains from a series of fourteen untreated cases. Sections were taken from twenty-four areas in each brain and stained by cresyl violet. Realizing that the histologic picture varies in different areas of the same brain, the twenty-four areas were studied from each brain and thus a fair idea of the situation could be obtained.

One striking feature of general paresis is the presence of plasma cells in the perivascular infiltrations and in the meninges. It is pretty generally accepted that plasmacytosis is essential for a diagnosis of general paresis. I show you a lantern slide picture from a case of untreated general paresis showing a small vessel surrounded by infiltration consistsing almost entirely of plasmacytes. This second picture from a case of general paresis in which the patient had received treatment shows some perivascular lymphocytic infiltration but no plasma cells. This is rather characteristic of the treated and untreated cases; namely, in the treated cases plasmacytes were comparatively rare as contrasted with the untreated cases. Sixteen of the twenty-seven treated cases showed practically no plasmacytic infiltration. Six showed a moderate amount, and five showed a considerable amount. Of the five that showed considerable plasmacytic infiltration two or three had received a fair amount of treatment and the other two had received very little. Contrasted with this finding were the findings in the untreated series of fourteen. Of these, nine showed a considerable degree of plasmacytic infiltration and five a moderate amount. None of them showed a slight infiltration as was found in the sixteen of the twenty-seven treated cases. The story concerning the perivascular lymphocytic infiltration is similar but not so marked. Twelve of the twenty-seven treated cases showed a slight infiltration, ten a moderate amount, while five showed considerable. It is worth noting that a number of the cases showed a fair amount of lymphocytic infiltration and slight plasma cell infiltration. In the series of fourteen untreated cases not one was relatively free from lymphocytic infiltration whereas nine showed a moderate perivascular lymphocytic infiltration and five showed a marked infiltration with lymphocytosis. A number of the brains had had the pia stripped before being obtained for examination. However, nineteen of the treated cases had the pia intact. Of these nine showed a slight pial infiltration, seven moderate, and three considerable. In eleven of the untreated cases it was possible to examine the meninges. Of these one showed slight pial infiltration, four moderate, and six considerable. As regards the pial infiltration, it is rather striking that three of the treated cases had a considerable amount of pial infiltration with lymphocytes. *A priori,* it would seem that the pial inflammation is readily amenable to antisyphilitic drugs, but histologic evidence in these cases points to the fact that there are cases in which the antisyphilitic remedy does not give satisfactory results even as regards the pial infiltration. As to the glia cell picture, there was no difference between the treated and untreated cases. One would feel that from the difference in the type of the plasma cell picture it is possible to make a fair estimate by a histologic examination as to whether the cases had received antisyphilitic treatment. We believe we are justified in drawing the conclusion that something has been accomplished by the treatment of cases of general paresis as shown by the histology, even though this may not have any clinical value.

DISCUSSION

DR. H. I. GOSLINE: One slide seemed to show a certain paucity of nerve cells. Have such areas in treated cases been compared with similar areas in

untreated cases? My reason for asking that is that I have noticed in carrying out this treatment that neo-arsphenamin will start a severe dermatitis and lead to cicatrization if one does not cover the fingers. Then again the cell count drops as a most constant and uniform occurrence. Does this arsenic act as a sort of searing process, killing off epithelial cells, and does the cured paretic patient get on because he has a sufficient number of nerve cells left to function although he has been injured?

Dr. Donald Gregg: What happened to the ten patients under treatment whose cases you reported a year ago?

Dr. Solomon: One of them has had a relapse.

Answering Dr. Gosline, I would say that we have studied these cases in a great deal more detail than I have attempted to present this evening. We have studied the glia cells, the white substance, the ventricular surfaces, the dropping out of cells and have also attempted to discover whether there is any relation to the age of the patient or with the duration of the process. I do not believe we will have any striking results. I cannot answer definitely why some patients die with a small amount of atrophy and some with a tremendous amount. As to the spinal fluid findings, we have had some rather disappointing results. Some of the cases in which the spinal fluid was practically free from cells during life show marked evidence of meningeal and perivascular infiltration. In one case particularly in which apparent cure had been accomplished, the serology was entirely negative, yet there was perivascular infiltration and meningeal involvement. Dr. McPherson asked me whether it was a good thing or a bad thing to lose one's plasma cells when one has paresis. I do not know. The fundamental study we should like to make would be on the amount of spirochetosis these brains show, but this is not possible at present owing to the technical difficulties in demonstrating spirochetes.

PHILADELPHIA NEUROLOGICAL SOCIETY

Regular Meeting, Nov. 25, 1921

George Wilson, M.D., *President, in the Chair*

A CASE OF BILATERAL EIGHTH NERVE PALSY, WITH PARTIAL RETURN OF FUNCTION AFTER TREATMENT. Dr. M. A. Burns.

This patient was again shown after the lapse of a year, because of the partial return of function after treatment. One year previously this man had been totally deaf in both ears. At the time of presentation he could hear with his right ear.

When treatment was begun the blood Wassermann reaction was ++++; the globulin was markedly increased; there were 104 lymphocytes to the cubic millimeter of spinal fluid and the Wassermann reaction of the spinal fluid was ++++. The blood Wassermann reaction now was ++; the spinal fluid Wassermann reaction was negative; the globulin was slightly increased and there were nine lymphocytes to the cubic millimeter of spinal fluid. Up to the time of presentation, the spinal cord had been drained thirty-five times; he had had forty intravenous injections of arsphenamin, as much as 50 grains (3.25 gm.) of potassium iodid every three hours and 2 drams (7.8 gm.) of mercurial ointment, daily.

Hearing began to return in the right ear in March, 1921, and when presented he could hear an ordinary conversation over the telephone.

DISCUSSION

DR. FRANCIS X. DERCUM: This man was practically stone deaf when I first saw him in the clinic, and the return of hearing is very interesting. The case is remarkable because of the bilateral character of the lesion.

DR. S. F. GILPIN: I have followed this case for a year with considerable interest because of the discussion which followed his presentation to the society a year ago. At that time the diagnosis made was syphilitic basal meningitis. Experience has shown that in neurosyphilis with an increased number of lymphocytes, the giving of intensive treatment accompanied by drainage of the cerebrospinal fluid produces results which cannot be obtained in any other manner.

A PATIENT WITH BULBAR PALSY AND AMYOTROPHIC LATERAL SCLEROSIS APPARENTLY FOLLOWING LETHARGIC ENCEPHALITIS. DR. S. F. GILPIN.

D. L., a white man, aged 31, married, a laborer by occupation, was admitted to the Jefferson Hospital, Oct. 31, 1921. The family and past history were negative, except that he was an eight months' baby and was delicate until the fourth year. The patient denied all venereal history. He had healthy children. The present trouble began with an attack of influenza and pneumonia in the epidemic of 1918. Following this for about four months, he had a great desire to sleep. He worked for one or two days a week but was weak and fell asleep whenever he was quiet. He had no pain, he did not vomit, and there were no eye symptoms. In the fall of 1919, he noticed weakness in his right arm, accompanied by wasting of the muscles, which soon extended to the left arm. The arms had gradually become worse until at present the patient could not feed or dress himself. The legs became involved about the same time as the arms, becoming weak and stiff. The speech disturbance came on gradually with the other symptoms.

Examination revealed marked wasting of all the muscles of the upper and lower extremities. The legs were not wasted to as great a degree as the arms, and the patient was still able to walk, though his gait was weak and spastic. His pupils were equal and reacted normally. He stood well with his eyes closed. His tongue was protruded with difficulty, was wrinkled and showed atrophy. His throat muscles were weak and he had difficulty in swallowing. He had almost no use of his arm or hands. He showed exaggerated knee jerks, a bilateral Babinski sign and ankle clonus. He had no loss of sensation, of sphincter control or of sexual power.

REPORT OF A CASE WITH UNUSUAL SYMPTOMS PROBABLY DUE TO INVOLVEMENT OF THE ANTERIOR HORNS AND THE LATERAL COLUMNS. DR. E. A. LEONARD.

History.—The patient was an ex-service man, aged 29, and unmarried. He was drafted in May, 1918, after two attempts at enlistment and he was discharged, October, 1918, on a surgeon's certificate of disability. The family and past medical history were negative. According to the patient's mother, at 16 months of age, his left arm became suddenly useless without any other symptoms, such as fever or vomiting. At the time of this sudden loss of

power in the left arm the boy was holding a potato when his hand suddenly relaxed and the potato fell from his grasp. There had been loss of power and wasting since that time. There was no history of pain.

Physical Examination.—The cranial nerves and pupils were normal. There was marked wasting of all muscles of the left upper arm and of the muscles over the chest, but not to such a degree as in the left arm. The wasted muscles were the seat of fibrillary twitchings. Both upper extremities were the same length; one was flaccid, but there was no failure of bony development. There was no disturbance of sensation. The reflexes in the left arm were present and showed slight exaggeration, considering the extreme degree of wasting. The reflexes in the right arm were about normal. The muscles of the legs showed some hypertonicity and no wasting. The knee jerks were exaggerated; the abdominal. plantar and cremasteric reflexes were normal and there was no ankle clonus. The electrical reactions, as tested by Dr. M. K. Myers, showed that the muscles of the left extremity had retained their faradic irritability; the biceps reacted normally to the galvanic current, but in the muscles of the left forearm an anodal closing contraction produced more reaction than the cathodal closing contraction.

DISCUSSION

Dr. S. F. Gilpin: The patient has no clonus or Babinski reflex, though his knee jerks are slightly active and he has had wasting of one arm for years. If he had, at 16 months of age, what appeared to be acute anterior poliomyelitis, would the patient's arm have developed in length as it has? One arm is as long as the other. The question arises as to whether there is a chronic anterior poliomyelitis which has developed after the acute attack or whether the present condition is the result of the acute poliomyelitis in childhood.

Dr. A. J. Ostheimer: I believe that the case is one of anterior poliomyelitis occurring in early childhood, on which there has now developed a chronic anterior poliomyelitis with the pathologic changes to a certain extent going over into the lateral tract. This whole subject was very thoroughly covered in an article by Dr. C. S. Potts, in 1903, published in the University of Pennsylvania Medical Bulletin. He reviewed the literature citing thirty-eight cases of early anterior poliomyelitis, which in later life gave rise to progressive muscular atrophy or amyotrophic lateral sclerosis.

CRURAL PARAPLEGIA OF CORTICAL ORIGIN. Dr. George Wilson.

History.—The patient, an ex-service man, aged 27, was wounded in the head by a shell fragment in France and was rendered unconscious for six days, after which he was paralyzed in both legs and had incontinence of urine and feces. He began to regain power in the legs eight months after he was wounded; and the control of the bowel and bladder returned at that time. His sexual power, which had been affected, also gave evidence of a revival at that time. He was able to walk, one year after the injury. He never had disturbance of pain, or of heat or cold sensations in the lower extremities.

Physical Examination.—The gait was spastic and was rendered somewhat bizzare because of a flaccid paralysis of both feet, which was practically complete. The rest of the lower extremities were weak but not paralyzed. Romberg's sign was not present. The deep reflexes of the lower extremities were exaggerated, with a bilateral Babinski sign but without ankle clonus. The

abdominal and cremasteric reflexes were normal. Except for slight disturbance of the sense of position and tactile localization in the toes of the left foot, there was no impairment of sensation.

The case was of interest from two points: first, because of a spastic paralysis of both legs due to injury of both leg centers, and, secondly, because of the paralyses of the bowels, bladder and sexual function due to cortical lesions. The case will be reported in detail with a case of cortical crural monoplegia.

DISCUSSION

DR. CHARLES K. MILLS: This case is interesting from the standpoint of cortical localization. While it is true, as Greenbaum and Sherrington and others following them have reported, that there are centers in the upper motor region, on the lateral or mesal side of the hemisphere near its edge, still we have comparatively few cases of disorder of the bowels, bladder and sexual functions from unilateral lesions of the cortex. The probable explanation of this is that for functions of this sort, centrally exhibited, as for other functions, more or less bilaterally expressed, the center in one hemisphere may function for both sides of the body, or for its central region in which are included the bowels, bladder and sexual organs.

UNUSUAL SEQUELAE OF EPIDEMIC ENCEPHALITIS (LETHARGICA). DR. ALFRED GORDON.

History.—A girl, aged 11, became ill in January, 1921, with a sore throat, marked tremor of the hands and a temperature of 99.5 F. The following day, the tremor became generalized and there occurred an attack which the parents called a convulsion. Two similar attacks occurred two days later. Her temperature rose to 101.5 F. and she became confused. On the fifth day, myoclonia was observed, which was diagnosed as chorea. From that time on, the patient became somnolent and her temperature rose to 102 F., with a pulse of 136, and the speech became indistinct. The knee jerks at that time could not be obtained. There was no cranial nerve involvement with the exception of a sluggish light reflex in the right eye. The condition remained stationary for several days. On the twentieth day some improvement was noticed. At that time, a lumbar puncture was made and the spinal fluid showed 34 lymphocytes per cubic millimeter. The Wassermann reaction was negative. The globulin test (Noguchi) was positive.

The patient showed slight improvement, but for many days complained of pain over the upper part of the spine. Twitching of the muscles of the body was evident and continuous; it was synchronous with her respiratory movements, which were quite rapid.

Examination.—Four months later, I examined the patient with these results: The arms were animated with regular rhythmic flexor movements, accompanied by supination of the forearms and flexion of the fingers. They were coincident with identical muscular contractions of the diaphragm, so that there was a synchronous movement of the upper abdomen and upper extremities. These movements were all quite rapid. The face presented spasmodic contractions confined to the muscles of the left side and only in its lower half. The facial spasm was not synchronous with the muscular contractions of the arms, but considerably more rapid. The patient held her head in a peculiar position, inclined forward at an acute angle. There was no fixation for the head and the trunk could be straightened without difficulty. The knee jerks

were exaggerated. The pupils were unequal, the left larger than the right. The grip of both hands was poor and the thenar and hypothenar elevations were flattened. The respiration was rapid and the heart beat was 144 per minute. The blood pressure was 118, systolic. All vegetative functions were normal. Mentality was good.

Comment.—This was probably a case of epidemic encephalitis (lethargic) in which the third, seventh and tenth nerves were involved. The facial spasm indicated an irritative lesion of the seventh nerve, only of one half of it, however, since only the lower half of the face was involved. There was also an irritative state of the last four segments of the cervical cord. The diaphragmatic contractions also indicated an irritation of the phrenic nerve which originates in the fourth cervical segment. The flattening of the thenar and hypothenar muscles suggested some involvement of the cells of the anterior cornua of the lower cervical segment. The irritative lesion was either in the roots of the nerves or else in the central portion of the cervical cord, although the cervical pain favored the former. The lesion was certainly partial since not all the muscles supplied by these nerves were involved.

CHICAGO NEUROLOGICAL SOCIETY

Regular Meeting, Dec. 21, 1921

C. F. READ, M.D., *President, in the Chair*

A CASE OF HABIT SPASM OF THE MOUTH AND TONGUE. DR. HUGH T. PATRICK.

History.—A temperamental woman, aged about 50, who had given public readings since childhood, had led a practically normal and uneventful life until November, 1919, when her husband died of heart trouble very suddenly, in her presence. She was greatly shocked. Following her husband's death, she was much depressed and very unhappy; she did not wish to see people so she went into the country and was quite solitary for a time. During this period, when urged to take nourishment, she said she felt as if she could not eat, but she did eat. There were no very striking symptoms until June, 1920. She then began to have difficulty in mastication, being unable to close the jaws. When she was about to eat her jaw would drop spasmodically and she would have trouble in closing it. This condition had persisted; but three months previously, it began to improve and at the time of presentation it was considerably better. In June, 1921, she began to have difficulty in articulation, which was rather complicated. First, there was difficulty with the jaw; the mouth opened very widely and she had difficulty in closing it. It remained wide open for several seconds before she could talk, and this spasmodic depression occurred at intervals as she proceeded. There was also difficulty with the lips and tongue, the tongue being moved about in a most irregular way, often protruding beyond the lips and momentarily being held rigid. The lips were less involved but were pouted and retracted in irregular but coordinated movements.

When the patient expected to drink or eat, there was generally a sudden opening of the mouth, and the jaw was held that way for a short time. There was no trouble in the act of deglutition; but in masticating there was dif-

ficulty in prompt motion of the jaw. The movements of the tongue were clumsy, slow and irregular. It did not respond promptly or properly to the morsel of food. Although speech was irregular and interrupted, articulation was good. When unnoticed the patient could take a drink of water without difficulty and could eat with little trouble. Like a patient with spasmodic torticollis, she had a "restraining gesture." When she pressed up the chin with her hand she could speak with more fluency, although if she was thoroughly rested she spoke much better. She also spoke better when she was very much absorbed or in earnest.

It was not an easy case to classify. There was a striking resemblance to spasmodic torticollis in the restraining gestures and in the way in which the depressed jaw was held fixed for a few moments. Many features were like those of tic, but a tic persists when the patient is entirely at rest; it is quite spontaneous. This patient's trouble appeared only when she talked or ate. In that respect it resembled an occupation neurosis. Although in eating the tongue moved about in the most erratic way and occasionally the jaw closed with a snap, the tongue was never bitten. Therein it resembled a tic. The more self-conscious she was, the worse the abnormal movements. The more automatic her action, the more nearly normal.

Comment.—I have not had time fully to investigate the etiology of this case; but a few interesting points have been ascertained. First, there is nothing to indicate that the patient has had lethargic encephalitis. Second, there are no signs whatever of organic disease. Third, following the loss of her husband, she not only had business and financial difficulties but some very perturbing emotional conflicts. Fourth, and most interesting, her husband died as they were about to retire for the night and had removed his false teeth concerning which he had always been abnormally sensitive. With this sensitiveness she had been most sympathetic. As soon as the immediate commotion of the sudden death had subsided, she put everybody out of the room and compelled herself to put the plate of false teeth in place and arrange the mouth as she thought it should be. But she was haunted by a fear that something might go wrong; and when the remains in the casket were exposed for view she requested a friend to see if "the mouth was right." Unfortunately, the friend reported that "the mouth was wrong," when it was too late to change it.

A CASE OF TABES DORSALIS SHOWING ATAXIA OF SUDDEN ONSET.

Dr. William H. Holmes.

The third series of the Index Catalogue of the Library of the Surgeon-General's Office, U. S. Army, issued in 1920, contains seventeen references to acute ataxia. Seven of the articles referred to were available to me. None of the articles consulted refer to acute ataxia in true tabes dorsalis; but doubtless many such cases could be found by extensive search. Textbooks on neurology mention that ataxia in tabes may appear suddenly and progress rapidly; but they are not very specific as to the period elapsing between an asymptomatic stage of the disease and a stage of complete paralytic ataxia. Maloney's monograph on tabes mentions several cases in which fatigue seemed to precipitate the ataxia. My case showed development of severe ataxia of the lower extremities in five days.

History.—The patient, aged 30, gave a negative history so far as the present condition was concerned. There had been a Neisserian infection ten years pre-

viously; syphilis was denied. He served in the Army during the recent war and was discharged in good physical condition. Prior to his marriage, two years previously, he drank heavily; afterward he drank on only a few occasions, the last time being about four weeks before the onset of the present trouble. At that time he drank about one-half pint (0.28 liter) of whisky. Three or four days after this episode, he suffered from sharp, shooting pains in the lower extremities which persisted for three or four days and then entirely disappeared. He had never had any difficulty in walking, nor paresthesias, visual disturbance, loss of sexual power, etc. For some months, however, he had noticed that urination was infrequent and that urine could be retained for twenty-four hours without discomfort.

Nov. 7, 1921, while following his occupation of salesman, he noticed numbness of both feet, legs, thighs and buttocks. The following day, the numbness was more pronounced and some difficulty in walking was experienced. The third day the subjective feeling of numbness and the ataxia were quite marked; although he was able to stand alone and to walk with slight assistance. November 11, the lower extremities felt quite dead and walking without assistance was impossible. He was first seen by his attending physician, November 12, at which time it required the assistance of two strong men to enable him to stand. In bed he was unaware of the position of his lower extremities. There was no pain in the legs nor tenderness on pressure, and no loss of sphincter control.

Examination. — November 13, the temperature was 98.6 F., the pulse 96. Results of examination of the nose, throat, heart, vessels and abdomen were negative. There was diffuse chronic bronchitis. The urine was negative. Blood counts and the Wassermann test were not made. The pupils were unequal in size, the left being slightly larger than the right. They were regular in outline and reacted promptly to light and accommodation. The ciliospinal reflex was normal; as were also the cranial nerves. There was no disturbance of speech. With the exception of the right biceps jerk, the tendon reflexes of the upper extremities could not be obtained. There was moderate ataxia, as evidenced by poor performance of the finger-to-finger and finger-to-nose tests and by a difference in the writing with the eyes open and closed. Sensation of all types and muscular power for all movements were apparently normal in the arms, except that deep pressure was not painful. Epigastric and upper and lower abdominal reflexes were brisk and equal. Sensation on the trunk was normal. Pressure on the testicles was not painful until the degree of pressure became extreme. Neither cremaster reflex could be obtained. Knee and Achilles jerks could not be obtained. There was normal plantar flexion. Both lower extremities were normally and equally powerful. Ataxia was extreme and was increased by closing the eyes. When the eyes were closed, the position of the extremities was unknown. Tactile sensation seemed perfectly normal and without tardy response. Pin prick was appreciated as painful over both extremities. Tactile discrimination was impaired. When two points were placed at right angles to the extremities, they were not correctly located unless separated by from 1 to 1½ inches (3.8 cm.). Bone vibration sense was decreased as compared with that of the upper extremities. Deep pressure was not painful. Only very large coarse movements of the great toes, ankles and knees were appreciated and then only apparently by reason of skin stretching. Standing alone or walking with the assistance of two men was not possible because of the severe ataxia. The spinal fluid was clear, contained 90 cells per cubic millimeter. Tests for albumin and

globulin were positive. The Wassermann reaction was strongly positive and the Lange curve was typically syphilitic. The diagnosis was tabes, with the occurrence of acute ataxia. Ataxic polyneuritis complicating tabes was considered but was eliminated because of the complete absence of pain and tenderness, the negative history, and the presence of normal muscular power. The electrical reactions were not studied.

Course.—During the month which has elapsed since the examination recorded above, he has had frequent attacks of lightning pains. There has been a steady improvement and he is now able to walk with the use of a cane. He has had no treatment other than complete rest and mercury by inunction.

DISCUSSION

DR. HUGH T. PATRICK: I think these acute exacerbations of tabes are among the most interesting things connected with the disease. They present some practical features. Any nonprogressive or slowly progressive case of tabes may have one of these acute exacerbations; but, in my opinion, tabes never begins that way. The next thing is that, acute and severe as these cases may be, they do not necessarily carry a bad prognosis. This is exemplified in this patient who a month ago could not walk at all and now walks fairly well with a cane, even without a cane. In some of the severe cases, it looks as though the patient would succumb, but I have never known of one of these acute cases being fatal, although of course this may have happened. Sometimes they do not improve very much but remain nonprogressive. Many years ago, long before the days of the lumbar puncture, I saw a marked case in a small coal dealer, whose business called for a good deal of physical activity. Within twenty-four hours he became completely paraplegic, with marked sensory blunting to the nipple level and retention of urine. The case looked very grave indeed. He remained practically paraplegic; but for years after that he went about his business in a chair propelled entirely by the arms. He regained control of his bladder; his health was good, and he was as successful a merchant after losing the use of his legs as before.

The moral is not to be discouraged or to allow the patient to be discouraged about the outcome. Some cases of tabes begin rapidly but rarely do they so continue. In a case seen yesterday, the first symptoms began about a year ago, with numbness in the feet. The patient is now quite ataxic, with marked sensory disturbance and the trouble is much more marked than one would expect it to be in a year; but I have no idea the case will continue to progress at that rate. Such cases generally run a rapid course up to a certain point and then come to a standstill for an indefinite period or progress very slowly, just as ordinary tabes.

DR. H. DOUGLAS SINGER: I recall a case in which the onset seemed to have been within one-half hour. I am not absolutely certain of the facts, for it is a long time since I saw the patient; but as I recall them, the man was out in a park sitting in a chair and when he tried to get up he was so ataxic he could not walk. He was unable to stand the next morning. It occurs to me that possibly a different kind of lesion takes place in such cases and that these acute ataxic disturbances should be compared with the acute muscular paralyses observed occasionally, which clear up—apparently neuritic attacks which involve only the motor fibers of some nerve. I wonder whether these acute ataxic disturbances are not somewhat of the same nature, with a different type of lesion from the more chronic ones of tabes itself.

INTRACRANIAL ANEURYSM. Dr. George W. Hall.

History.—A man, aged 45, an office worker, with no history of previous illness except whooping cough at 9 years, had been deaf in the left ear since childhood with no definite history of the cause. The onset of the present illness occurred in the latter part of 1918, with headache, dizziness and vomiting, accompanied by numbness in the right half of the tongue, numbness and burning sensation on the right side of the face, weakness and numbness in the right arm and weakness in the right leg, followed by a tendency to walk to the right. There was a history of diplopia at intervals for several days after the attack. A similar attack occurred in the latter part of 1919 and a third attack in June, 1920. The last attack was very severe and was associated with severe headache on the right. In June, 1921, another severe attack came on suddenly, associated with severe headache, dizziness and vomiting. The vomiting was projectile. After this attack, he again noticed the tendency to go to the right, accompanied by ataxia of the right arm and leg.

Examination.—The urine was negative, except for a few hyaline and granular casts and a trace of albumin. The blood Wassermann reaction was negative. The blood pressure was 180, systolic. The palpebral fissure on the right was narrower than on the left; the right pupil was smaller than the left. Analgesia was noted over the distribution of the fifth nerve; no bruit was heard on auscultation of the skull. A roentgenogram of the skull was negative.

DISCUSSION

Dr. Ralph C. Hamill: Were there any subjective head noises?

Dr. Hugh T. Patrick: How did you make the diagnosis of aneurysm?

Dr. Hall: The diagnosis of aneurysm was based on the presence of arteriosclerosis, recurrent attacks of a certain definite syndrome, and an apparently good condition between the attacks. These were associated with unilateral headache.

Dr. Harold N. Moyer: Why could it not be just an ordinary cerebellar tumor?

Dr. Hall: As I have just stated, the symptoms subsided between attacks and the attacks were rather apoplectiform in character; whereas in cerebellar tumor, extending over three years, there would be more positive evidences of a tumor and the symptoms would not subside as in this case. Also, the optic disks were normal, excepting slight tortuosity of the vessels.

Dr. Hugh T. Patrick: If we are to make a diagnosis of intracranial aneurysm on this syndrome I believe we shall make it oftener than we have in the past. Brain tumors may cause these recurrent attacks. Why might not an aneurysm be a large thing by this time, like any other growth?

Dr. Hall: I am glad Dr. Patrick brought up the questions he did regarding the diagnosis because Dr. Wells will cover a great deal of the literature. I should like to cite one or two authors: Wichern (*Deutsch. Ztschr. f. Nervenh.* **44**:220, 1912) states that in fifteen of twenty-two necropsies there was evidence of previous leakage or rupture of the aneurysm on one or more occasions. He also calls attention to the unilateral headache accompanied by nausea, giddiness and vomiting and speaks of the sudden exacerbations with apoplectic onset of symptoms followed by varying periods in which no fresh developments appear. This in turn is followed by second, third and even fourth seizures. Multiple seizures were present in more than 31 per cent. of his cases. Beadles (*Brain*, 1907) states that the diagnosis of intracranial

aneurysm is very difficult; but that if there is any one sign deserving special attention it is the intermittent character of the symptoms. Unruptured aneurysms usually are symptomless.

INTRACRANIAL ANEURYSM OF THE VERTEBRAL ARTERY.

Dr. H. Gideon Wells (by invitation).

This article appears as an original communication in this issue, page 311.

DISCUSSION

Dr. Peter Bassoe: In regard to the clinical diagnosis, it would seem to me that a bony groove such as Dr. Wells described might have been detected on stereoscopic roentgen-ray examination; and when the symptoms are suggestive, as in the case of Dr. Hall, this might help in the localization. Of course, a tumor may erode bone just as well.

Dr. Rogers and I recently saw a patient, a business man, aged 52, who had lost his left eye when he was 14 years old, and who, some years ago, had an attack in which he lost himself in the street, after which he was confused for a few hours, remaining in bed for a week. He had had a good deal of headache during the past five years, as well as attacks of dizziness. Three years before, he had had an attack, following which he dragged the right foot. Five days before I saw him, he had been out fishing on a hot day. He came into the boat house and had a severe attack of vertigo; it seemed to him that the boat house turned around. The next day his speech was thick; he could not swallow, could not walk alone and had spasms of the right arm and leg. The following day there were spasms of the right arm, and two days before I saw him, the third day after the dizzy attack, he had a general convulsion during which he was unconscious and had involuntary micturition. He had improved somewhat when I saw him.

The chief findings, when I saw him, were a bilateral Babinski sign; increased knee jerks, the left greater than the right; absence of abdominal reflexes, and arm reflexes, a little increased on the left. There was impairment of pain and temperature sense on the left side, not involving the face; ataxia of the right hand and a subjective burning sensation in the right side of the face, and no impairment of sensation on the right side. Speech was a little thick and he dragged the right leg in walking. The eye grounds were normal. There was no increase of cells in the spinal fluid; but there was a positive globulin test and a slight Lange reaction. The Wassermann reaction was negative.

Was it a brain tumor or a vascular lesion? My opinion was that he had a vascular lesion and I thought at the time that there possibly was a thrombosis of the left vertebral artery. The symptoms were complex and in part resembled those of a thrombosis of the left posterior inferior cerebellar artery, which is the principal branch of the vertebral. After some weeks, the patient grew worse and was taken to Dr. Walter E. Dandy in Baltimore, who kindly informed me that he performed an exploratory operation and discovered a large aneurysm of the basilar artery and great dilatation of the left vertebral artery. Later, these findings were confirmed at necropsy. There was considerable softening of the pons and medulla. Hence, the symptoms were probably the result of circulatory disturbance rather than of mechanical pressure by the aneurysm.

DISCUSSION

Dr. Arthur W. Rogers: Subsequent to the time Dr. Bassoe saw the patient, he improved so much that he went out on the lake fishing again. At 4 o'clock one morning, I was called and found him in much the same condition in which I had seen him four or five weeks previously. He was in coma; and for the first time I discovered noticeable eye symptoms. The internal rectus had become paralyzed and the good eye turned far out to the temporal side. Within four or five hours, consciousness returned and an oculist found nothing but the paralysis of the internal rectus. This ocular paralysis was on the side opposite the anesthesia.

Dr. H. Gideon Wells: Regarding help from roentgen-ray examination, I imagine this would ordinarily be of little service because Dr. Bassoe's case and this one are about the only aneurysms described in the literature that were big enough. I think Beadles, in his report meant miliary aneurysms. An aneurysm as large as this one would produce symptoms anywhere. I think the reason it reached such a size was that it occurred when the man was young, and there was enough depression produced at the base of the skull to permit its reaching a large size without producing fatal pressure effects.

Dr. Hugh T. Patrick: Another indication that Dr. Wells' case was very slow in developing was the absence of cranial nerve paralyses. From the location of the tumor, there must have been such palsies if there had been a rapid growth.

I entirely agree with Dr. Bassoe's diagnosis in his case. The patient may have had some symptoms due to the aneurysm but most of them undoubtedly were due to thrombosis in the pons.

Perhaps I may be allowed to add that twice I have ventured a diagnosis of intracranial aneurysm, neither case proved by necropsy. I based my diagnosis on more or less indefinite symptoms of tumor with tinnitus, synchronous with the pulse and a bruit audible to the examiner. The stereoscope placed close to the ear enabled one to hear a bruit. I had one other case in which there was very definite tinnitus synchronous with the pulse, not audible to the examiner and partial paralysis of the seventh nerve. I made a diagnosis of tumor, not aneurysm. Dr. Fenger operated. I think the symptoms in that case nowadays would have led to the diagnosis of cerebellopontile angle tumor, but it proved to be a sarcoma of the petrous portion of the temporal bone.

THE PATHWAY FOR VISCERAL AFFERENT IMPULSES WITHIN THE SPINAL CORD.* Dr. Loyal E. Davis.

Practically nothing can be definitely stated in regard to the course of visceral afferent impulses through the sympathetic system, the cerebrospinal nerves and their connections in the central nervous system.

Several cats were experimented on, and vasomotor responses were obtained from stimulation of the thoracic sympathetic nerve. Stimulations of the same nerve were effected in cats on which various lesions of the spinal cord had been produced at the second dorsal segment.

Dorsal hemisection of the cord which destroyed both posterior funiculi and posterior gray columns had no effect on pressor responses obtained from the thoracic sympathetic nerve. On the contrary, this type of lesion very defi-

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nitely prevented a pressor response from stimulation of the sciatic nerve. This fact alone, therefore, rules out the posterior funiculi and any part of the posterior gray columns as the pathway for the conduction of this type of visceral afferent impulse. This is in contradistinction to the findings of Ranson and von Hess, working with somatic nerves, since they found the pressor pathway within the cord to be situated in the apexes of the posterior horns.

Neither does complete lateral hemisection cause a disappearance of the pressor response to thoracic sympathetic stimulation. This is true whether the nerve on the side of the lesion or on the opposite side is stimulated. This strongly suggests that whatever the pathway within the cord, it obviously must be one which ascends partly on the homolateral and partly on the contralateral side. Section of the lateral funiculi, including a portion of one of the anterior gray columns, has resulted in a slight decrease in the height of the pressor response; but the characteristic rise still remains.

A lesion of the anterior funiculi had no influence on the blood pressure curve. The only lesion produced which destroyed this pressor response from stimulation of the thoracic sympathetic nerve was a complete transverse section of the spinal cord.

It is reasonable to presume, therefore, that the impulses arising from the viscera, transmitted through the thoracic sympathetic nerve and giving rise to vasomotor and respiratory responses, are conducted upward by relays of short spinal paths with synapses in the gray matter of the spinal cord. If this be true, they resemble fibers which convey pain sensation in the cat from the periphery. Since complete transection of the spinal cord at the level of the second thoracic segment eliminated the pressor response, it is obvious that the reflex arc is not complete within the spinal cord and that the visceral afferent impulses must reach the bulbar vasomotor center before they can become effective in producing a pressor response.

DISCUSSION

DR. S. W. RANSON: There has been a great deal of work done in tracing the afferent somatic impulses through the spinal cord. But so far as I am able to learn, the experiments which Dr. Davis has just reported represent the first attempt to trace the course of visceral afferent impulses through the cord. It was theoretically possible that they could act directly on the spinal vasomotor centers, producing their effect through the cord. His results have shown, however, that this is not the case. These pressor impulses travel through the cord to the medulla oblongata and produce their effect through the bulbar vasoconstrictor center. This is also true of the somatic pressor impulses; but the visceral pressor path and the somatic pressor path are not the same. The somatic path is sharply localized in the apex of the posterior horn and in the tract of Lissauer, while the visceral path, as worked out in Dr. Davis' experiments, is obviously widely scattered through the cross section of the cord.

The study of visceral impulses is a new and largely unexplored field and a great deal of credit should go to Dr. Davis for making a distinct contribution to one of the underlying problems in this difficult field.

CORRECTION

In the title of the abstract of Dr. Cushing's article which appeared on page 263 of the February issue, the word temporary should have been temporal.

Book Reviews

BENIGN STUPORS. A Study of a New Manic-Depressive Reaction Type. By AUGUST HOCH, M.D. Cloth. Price, $2.50. Pp. 284. New York: The Macmillan Company, 1921.

The perusal of August Hoch's final work makes one doubly regret the untimely death of the author.

In this volume there is again evidence of the clinical acuity, the clear thinking, the constant searching beneath the surface which made him one of the acknowledged master minds of American psychiatry. It is particularly refreshing to note the full presentation of case histories and their keen analysis; the close insistence on the objective and subjective phenomena of the psychosis; the careful clinical observations and the freedom from glittering generalities. The study was not wholly completed by Hoch, and MacCurdy is to be commended for having edited the notes and brought them to light in their present form.

The monograph was prompted by Kirby's well-known observation that stupor in mental disease did not necessarily constitute a dementia praecox criterion. As the subtitle indicates, a benign stupor is a manic-depressive reaction type, and may be viewed as a recoverable psychosis which is characterized by inactivity, apathy, negativism, catalepsy, and a disturbance or abeyance of the intellectual functions. Partial stupors differ in degree but not in kind. The inactivity is not absolute, there is poverty of affect instead of affectlessness; negativism is less prominent. However, the intellectual processes are almost as markedly involved as in deep stupor. In patients in whom for an instant the curtain of apathy was lifted by a suicidal attempt there was also a brief modification from time to time of the other cardinal symptoms—thus revealing their close association and interdependence.

Probably the most valuable portion of Hoch's work is to be found in his study of the principal phenomena of the stupor not only by direct observation, but also in the light of the retrospective accounts of the patients; thus, we discover that intellectual functioning is of a primitive larval order and there is almost total amnesia both for external events and internal thinking. Comprehension and ideation are shallow, largely on account of the marked defect of attention. The ideational content of the stupor may be placed in a surprisingly narrow compass. In thirty-six consecutive patients, ideas of death were found in all but one; they were most common in the immediate prestuporous period. One-fourth of the patients gave a retrospective account of being dead, and the death concept is apparently often a clear simile for rebirth. "Naked came I from my mother's womb and naked shall I return thither." Apathy is the most important stupor sign. It is not at all the ultimate expression of depression and profound retardation, but constitutes an actual loss of affect and is as distinct a mood entity as are elation, sorrow, or anxiety. Inactivity is closely connected with apathy and, in fact, is its physical concomitant. It may be practically a total reaction, affecting even winking, swallowing and the sphincteric control. Retrospectively, one half of the patients spoke of feeling dead, numb, or drugged, and about 17 per cent. referred the phenomenon to external agencies. Catalepsy does not appear when the degree of inactivity

is extreme, and it may be the result of a deadlock between suggestibility and opposition, both being equally strong. In any event, the indefinite maintenance of an awkward posture is surely a manifestation of preservation. Retrospectively, catalepsy often was recalled as an hypnotic influence.

The chapter on the "Physical Manifestations of Stupor" struck a somewhat discordant note. The observations were few and incomplete and are in regrettable contrast to the completeness of the psychologic aspects. For instance, blood pressure was read in only two patients, and blood examinations made in only five! Fever was present in twenty-eight of thirty-five patients. It is interpreted as a result of a failure of heat loss function, which is dependent on an imbalance in the involuntary nervous system, which in turn is due to insufficient circulating epinephrin which finally is traced to the apathy. Neither this ingenious explanation nor the attempt to deny these stupors even the most remote kinship with organic reactions are at all satisfying or convincing. It is unfortunate that an attempt was made to draw conclusions from premises constructed of insufficient and incomplete data.

Psychologically, a benign stupor is an example of the simplest and most complete regression. The level of the regression is an index of the depth of the stupor. Often it reaches an infantile stage as evidenced by masturbation, soiling and wetting. In this deepest form it is a dramatization of the death idea.

The chief points of differential diagnostic interest concern malignant or dementia praecox stupors. In these there is no apathy, but an inappropriate affect, the attention and interest are not unable to function but are redirected to primitive fantastic thoughts with a consequent perversion of energy and emotion as evidenced by giggling, rage, etc. Finally, the death motif is absent. Retarded depressions are clearly different. There are physical concomitants of emotional life to be seen in the facies and bearing. Furthermore, while the patient is morbidly aware of the difficulty in thinking and slowness, he can when urged often perform tests successfully. In perplexed states there is too much affect and not too little. In absorbed manias the sequence of the psychotic history furnishes a valuable guide.

Treatment of benign stupor must for the present be confined to constant observation, careful nursing, and feeding, and to stimulation to effort in every direction.

Hoch's monograph clearly measures the enormous step which has been taken beyond the restrictions of a narrow and sterile descriptive psychiatry. It is indeed sad that this must be the final message from the pen of such a remarkable and brilliant clinician.

ATLAS FOR ELECTRO-DIAGNOSIS AND THERAPEUTICS. By F. MIRAMOND DE LAROQUETTE, M.D., Medical Director and Chief of the Electro-Radiologic Service of North Africa, Algiers. Translated by Mary Gregson Cheetham. Price, $4.50. Pp. 180. New York: Paul B. Hoeber, 1921.

This book contains little in charts or text that cannot be found on the bookshelf of any physician. The anatomic and physiologic discussion of the nervous and muscular systems, the joints, and the skin are quite elementary and of little value to the practitioner; unfortunately, they are also ambiguous and inadequate, and so would be of little value to one not already acquainted with the subject. For example (page 2), "Psycho-Sensory Motor Paths.—The psycho-sensory motor paths, or paths of conscious sensibility and voluntary

motion, have the same sensory and motor peripheral motor neurons and the same peripheral conductors as the reflex paths, but they present besides medullary and encephalic paths more or less complicated. They are branches of the reflex arc, which comprise the multiple stages, crossings and spinal, bulbar, cerebellar and cerebral connections which all end in the grey cortical matter of the brain. All nervous actions are, moreover, reflex, for they have all at least one stage of reflexion in the grey matter (Laulanié)." Again (pages 7 and 8), "Motor Fasciculi: Direct Pyramidal Fasciculus.—Middle anterior portion of the spinal cord: comes from the cerebral cortex on the same side, forms connections in the corpus striatum, traverses the medulla without crossing, and ends in the anterior horns of the opposite side. Voluntary motion. Crossed Pyramidal Fasciculus.—Postero-internal portion of the lateral cord: comes from the cerebral cortex of the opposite side, and ends in the cells of the anterior horns of the same side. Voluntary stimuli and motor coordination. The other fasciculi composing the white matter of the spinal cord and brain are formed of commissural fibers, with a tract more or less long, which bind the right and left portions of the different stages of the cerebrospinal column."

Ninety pages, consisting mostly of plates, are devoted to the location of motor points and their corresponding muscles, each of which is named in the text. The origin, insertion, action, innervation, motor points, and N. C. Con. (negative closing contraction), and P. C. Con. (positive closing contraction), with one value for each, are given.[a] A brief discussion covering the normal excitability of nerves and muscles is deferred to this point. Five tables, showing the electrical reactions in three cases of gunshot injury, one case of sciatic rheumatism, and one case of general neuritis, are interesting.

Sixty-one pages are devoted to a description of the various joints; the amplitude of movement, expressed in degrees, being given for each.

The last chapter of two and one-half pages covers the anatomy and the physiology of the skin. The inclusion of the word "therapeutics" in the title of the book is not only unnecessary but also misleading.

GUIDES FOR HISTORY TAKING AND CLINICAL EXAMINATION OF PSYCHIATRIC CASES. Edited by GEORGE H. KIRBY, M.D., Director, New York State Psychiatric Institute. Published by the New York State Hospital Commission, Albany, N. Y. Pp. 83. Utica, N. Y.: State Hospitals Press, 1921.

This book can be heartily recommended; it fills a real need of students and practitioners entering on the practical study of psychiatry. It is essentially a revision and elaboration of the outline prepared several years ago by Dr. Adolf Meyer for use in the New York State Hospitals, with well selected additions, especially for the study of personality and endocrine disturbances. In spite of the somewhat appalling appearance of length and detail, the guides will be found eminently practical and sufficiently elastic to permit adjustment to the needs of the individual case. The danger of losing individuality and developing a stereotyped phraseology, inherent in all schematic plans for examination, has been fully recognized and guarded against by repeated warnings in the text. An examination made with the intelligent use of the outlines furnished will provide the facts needed, not only for classification but also for the far more important questions of treatment, prognosis and etiology.

With regard to details, it might be suggested that the section dealing with the history of onset and development of the psychosis is disproportionately

brief in comparison with its fundamental importance. The omission of all reference to clinical laboratory investigations, except in the chapter dealing with endocrine disturbances, is striking, though perhaps justifiable. Typographic errors are somewhat numerous; the phrase, "may refuse to speak or answer," on page 62 should be corrected to "may not speak or answer." The paper tears easily and the volume is too large for the pocket, both of which are unfortunate in view of the manner in which the book will be used.